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Journal of Occupational Medicine

EVALUATION OF Occupational Disease Reporting in California

Beth M. Berkov, B.A., and Goldy D. Kleinman, M.A., Berkeley, Calif.; Jean E. Powers, A.B., San Francisco, Calif.; and Victoria M. Trasko, A.B., Washington, D. C.

SINCE 1949, the California Department of Public Health has been publishing statistics on occupational diseases in California. These data are generally acknowledged as the best available on the occurrence of occupational diseases for any state in the United States. The source of the statistics is the "Doctor's First Report of Work Injury," which serves as a means of notifying insurance companies and the state that the doctor has started treatment for what he considers to be an occupational accident or disease.

Owing to the preliminary nature of the doctor's first report, questions are raised from time to time reflecting upon the accuracy, quality, and completeness of reporting of occupational diseases. One objection often raised is that the reports have to be made too soon, and, as a result, the diagnosis is sometimes tentative and may not reflect the doctor's final opinion. In addition, the hazard involved is not always known at the time of the first examination; nor can the doctor always be sure that the illness is work-connected. Also of concern is the matter of whether the statistics of occupational diseases represent an overestimate or underestimate of the occupational disease problem.

Mrs. Berkov and Mrs. Kleinman are Public Health Analysts, Bureau of Occupational Health, California Department of Public Health; Mrs. Powers is Head, Work Injury Statistics Section, Division of Labor Statistics and Research, California Department of Industrial Relations; and Miss Trasko is Program Advisor, Division of Occupational Health, Public Health Service, United States Department of Health, Education and Welfare.

Submitted for publication Nov. 15, 1960.

This evaluation study of occupational disease reporting was undertaken to determine to what extent the suspected limitations are real and what effect, if any, they may have on the validity of occupational disease statistics.

The study was conducted by the California Department of Public Health in cooperation with the Division of Labor Statistics and Research, California Department of Industrial Relations. Assistance in planning and conducting the study was furnished by the Occupational Health Program, United States Public Health Service. The findings of this study are presented following a brief description of the California legal reporting requirements.

California Work Injury Reporting Requirements

California's Labor Code requires doctors and employers to file with the Division of Labor Statistics and Research, Department of Industrial Relations, reports of every injury to an employee arising out of or in the course of employment which causes disability beyond the day of the injury or requires medical treatment. The employer must file his report within five days after the injury occurs and the doctor who treats the injured worker within five days after first treatment.

The reporting requirements apply to well over 4,500,000 workers covered by the California Workmen's Compensation Act.

By interagency agreement, the Department of Industrial Relations refers all doctor reports concerning occupational diseases to the California Department of Public Health for statistical analysis and for carrying out its part

of the total program for the prevention and control of occupational diseases. Approximately 20,000 such reports are received each year. The reports serve as the source for annual statistical studies of the incidence of occupational diseases and for special studies such as those in connection with agricultural chemical illness, lead poisoning, and dermatitis. A unique feature of the statistics is that both disabling and nondisabling occupational diseases are analyzed. A "disabling condition" is defined as one causing absence from work beyond the day of occurrence.

Employer reports of disabling work injuries, including occupational disease, provide the source for statistics compiled by the Division of Labor Statistics and Research, California Department of Industrial Relations. Monthly, annual, and special analyses of work injuries are published by the Division.

Scope of Study

The study was limited to the evaluation of reporting characteristics having some bearing on the reliability of occupational disease statistics being currently compiled by the California Department of Public Health. October, 1958, was selected as the study period. October is a month when a relatively large number of occupational diseases are reported. The distribution of these cases is generally similar in diagnosis and hazard to the total occupational

ABSTRACT OF DOCTOR'S FIRST REPORT OF WORK INJURY

Do not write in this space

1. **EMPLOYER** .....
2. Address (No., St. and City) .....
3. Business (Manufacturing shoes, building construction, retailing men's clothes, etc.) .....

4. **EMPLOYEE** (First name, middle initial, last name) .....
5. Address (No., St. and City) .....
6. Occupation ..... Age ..... Sex .....
7. Date injured .....
8. Date of your first examination .....

11. **ACCIDENT OR EXPOSURE:** Did employee notify employer of this injury? ..... Employee's statement of cause of injury or illness:

12. **NATURE AND EXTENT OF INJURY OR DISEASE** (Include all objective findings, subjective complaints, and diagnoses. If occupational disease state date of onset, occupational history, and exposures.)

13. X-rays: By whom taken? (State if none) _____
Findings:

PLEASE COMPLETE THIS SECTION, FOLD, SEAL AND MAIL *(No Postage Required)*

1. Was this patient seen again after your first examination? ☐ Yes ☐ No

2. Do you have any further information about the accident or exposure in addition to the patient's statement above?
☐ Yes ☐ No If yes, please explain

3. Is the diagnosis above your final diagnosis? ☐ Yes ☐ No If no, what was the final diagnosis?

4. Is your present opinion that the condition was work connected? ☐ Yes ☐ No ☐ Undecided

Signature ..... Date .....

Fig. 1

disease reports for the entire year. A total of 2,331 cases of disabling and nondisabling occupational diseases were reported by doctors and employers as occurring during October, 1958.

One phase of the study dealt with obtaining answers to the often asked question, "How accurate is the information on the doctor's first report of occupational disease?" A follow-up questionnaire was mailed to reporting doctors to ascertain whether after filing the first report they had additional information on diagnosis, hazard, or disability and whether the condition was work-connected.

Another phase was concerned with determining the completeness of reporting occupational diseases by doctors compared with employers. A special match was made of doctor and employer reports of both disabling and nondisabling occupational disease to learn to what extent they report the same cases. As another measure of completeness of reporting, a catalogue was kept of doctors submitting at least one report of occupational disease during a six-month period to determine how large a group of doctors participate in reporting.

A third phase of the evaluation study consisted of comparing disability, diagnosis, and hazard or cause of illness as reported by doctors and employers on matched reports.

Although the desirability of doing so was recognized, it was not feasible to employ more refined evaluation methods such as follow-up through industrial hygiene surveys and personal interviews with reporting and nonreporting physicians. It is quite likely that such methods would result in a more accurate picture of the actual incidence of occupational diseases as contrasted with reported cases, but the undertaking of the task was beyond the scope of the study.

Follow-Up of Doctor First Reports

Follow-up questionnaires (Fig. 1) were mailed to reporting doctors approximately six weeks after the receipt of the first report. In order to identify the case, the inquiry form included an abstract of the information given on the "Doctor's First Report of Work Injury." A second inquiry was sent if the doctor failed to reply within one month. Questionnaires were not mailed for 349 cases of poison oak dermatitis reported during the study period because there would be little question about diagnosis of this condition.

QUESTION 1—*Was this patient seen again after your first examination?* (Fig. 2)

Answer	No.	%
Yes	944	69
No	409	30
No reply	20	1
Total	1,373	100

QUESTION 2—*Do you have further information about the accident or exposure?* (Fig. 3)

Answer	No.	%
Further information	49	4
Case not work-connected	84	6
Essentially no further information	1,233	90
No reply	7	....
Total	1,373	100

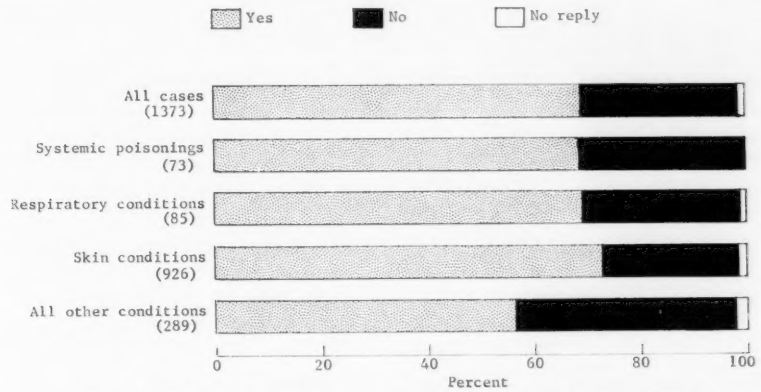

Fig. 2—Was this patient seen again after first examination?

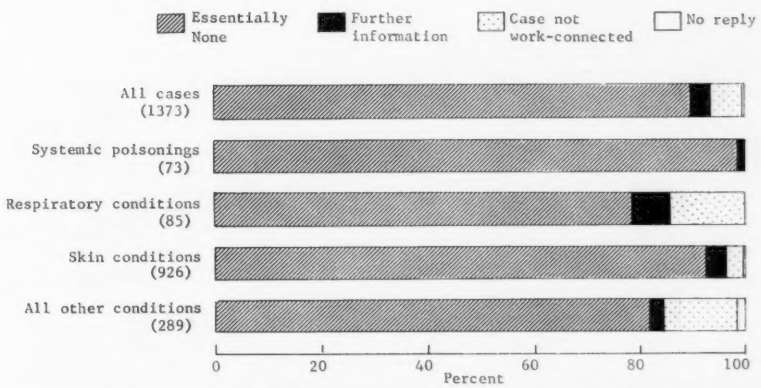

Fig. 3—Do you have further information about the accident or exposure?

A total of 1,415 questionnaires were mailed to doctors, and replies were received from 1,373, or 97%. While the high rate of response indicated interest in the study, the replies revealed that, on the whole, doctors had little additional information on the four questions asked of them.

Results of the replies to these questions follow and are illustrated for diagnostic groups in Fig. 2 to 5.

Replies to the question "Was this patient seen again after your first examination?" revealed that nearly 7 out of 10 patients were seen again after the first examination. It would appear that there is usually ample opportunity to question the worker further about his job or to seek information about the circumstances of the alleged condition when necessary.

As shown in Fig. 2, patients most frequently seen again were those with skin conditions (73%). The proportion of patients with systemic poisoning or respiratory conditions seen again was the same as that for all diagnoses (69%). Patients whose diagnoses were classified under "all other conditions" were asked to return less frequently (56%). "All other conditions" includes ear conditions, heat exhaustion, infective and parasitic diseases, and a relatively large number of vaguely stated conditions.

QUESTION 3—*Is the first diagnosis your final diagnosis?* (Fig. 4)

Answer	No.	%
Final diagnosis changed	13	1
Case not work-connected	84	6
Essentially no change	1,269	93
No reply	7	....
Total	1,373	100

QUESTION 4—*Is your present opinion that the condition was work-connected?* (Fig. 5)

Answer	No.	%
Yes	1,120	82
No	84	6
Undecided	129	9
No reply	40	3
Total	1,373	100

At the time he prepares his first report of an occupational disease, the doctor usually relies on what the patient tells him about the hazard and/or exposure that allegedly made the patient ill. Among all doctor reports processed annually, information on cause of illness is not specified for 12%. In undertaking a follow-up of reports, it was hoped that further information on cause of illness would be obtained. This did not turn out to be the case. In only 4% of the replies was the additional information sufficiently different from that on the first report to change the hazard classification code. Another 6% of the cases were found to be not work-connected. To what extent this decision was the result of further information obtained by the doctor or represents a revised judgment is not known. For most cases (90%), however, the doctor had no additional significant information about the cause of illness, even when the original facts were vaguely or incompletely stated.

Replies to the question "Is the first diagnosis your final

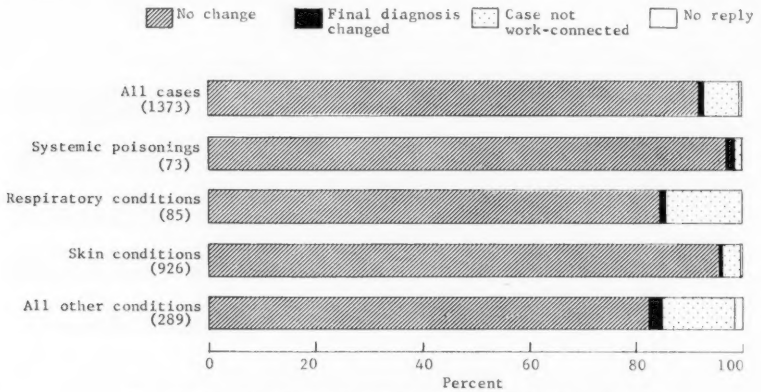

Fig. 4—Is the first diagnosis your final diagnosis?

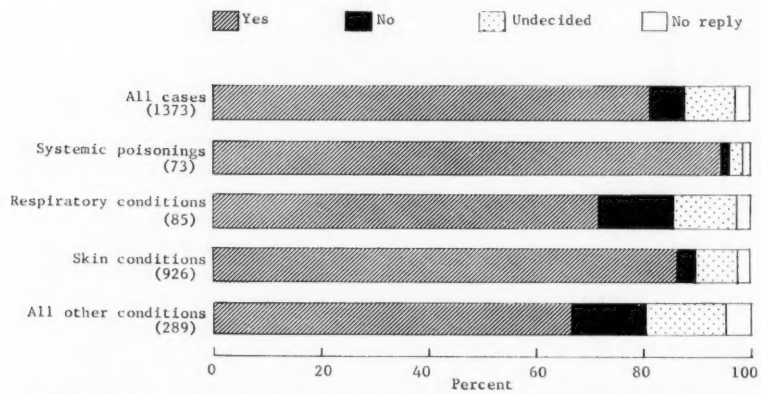

Fig. 5—Is your present opinion that the condition was work-connected?

Table I—Cases of Occupational Disease Reported by Doctors, Employers, or Both, California, October, 1958

Reported by	Total		Disabling		Nondisabling	
	No.	%	No.	%	No.	%
Total cases of occupational disease	2,331	100.0	749*	100.0	1,582	100.0
Both doctor and employer	1,592	68.3	569*	76.0	1,023	64.7
Doctor only (employer report missing)	423	18.1	87	11.6	336	21.2
Employer only (doctor report missing)	316	13.6	93	12.4	223	14.1

*Reported disabling by doctor or employer or both

diagnosis?" showed that the diagnosis made on the first report was the final diagnosis in 93% of the cases. Except for the 6% of cases later considered to be not work-connected, changes in diagnosis occurred infrequently (1%).

In 82% of the cases, the doctor confirmed his initial opinion that the condition was work-connected. He changed his first opinion in 6% of the cases and was undecided in 9%.

When diagnostic groups were considered separately (Fig. 5), systemic poisonings showed the highest proportion (95%) confirmed as work-connected and skin conditions next highest (86%). The proportion for respiratory conditions was 72% and for the group "all other conditions," 66%.

Completeness of Reporting

In this study, completeness of reporting was evaluated only to the extent of comparing reporting by doctors and employers. Reports were matched on the basis of name of company, name of employee, and date of injury, which involved reviewing about 50,000 records. Some matches undoubtedly were missed in spite of extensive searching.

A total of 2,331 cases of occupational disease was

Table II—Diagnosis and Hazard as Reported in Matched Doctor and Employer Reports of Occupational Disease, California, October, 1958

Diagnosis and Hazard	Doctor's First Report of Work Injury		Employer's Report of Industrial Injury	
	No.	%*	No.	%*
Total	1,592	100.0	1,592	100.0
Systemic effects of poison	75	4.7	48	3.0
Lead and its compounds	2	0.1	1	0.1
Metal fume fever	1	0.1	1	0.1
Other metals	3	0.2	3	0.2
Halogenated hydrocarbon solvents	4	0.3	1	0.1
Other solvents	3	0.2	3	0.2
Carbon monoxide	4	0.3	2	0.1
Other gases and vapors	14	0.9	8	0.5
Agricultural chemicals	37	2.3	26	1.6
Other chemicals	7	0.4	3	0.2
Respiratory conditions	79	5.0	83	5.2
Dusts	15	0.9	15	0.9
Gases	4	0.3	7	0.4
Metals and their compounds	3	0.2	3	0.2
Chemicals	37	2.3	35	2.2
Infectious agents	3	0.2	2	0.1
Other agents	1	0.1	3	0.2
Unspecified agents	16	1.0	18	1.1
Skin conditions other than poison oak	773	48.6	755	47.4
Agricultural chemicals	42	2.6	27	1.7
Nonedible plants and woods	21	1.3	22	1.4
Food products	95	6.0	102	6.4
Paints, varnishes, lacquers and sealers	11	0.7	10	0.6
Petroleum products (cutting fluids, greases, lubricants)	31	1.9	36	2.3
Soaps, detergents, and related compounds	59	3.7	44	2.8
Solvents	33	2.1	30	1.9
Acids and alkalis	18	1.1	11	0.7
Cement	23	1.4	22	1.4
Plastics and resins	33	2.1	37	2.3
Fiberglass and other inorganic dusts	37	2.3	35	2.2
Chromium	2	0.1	2	0.1
Other metals and their compounds	7	0.4	10	0.6
Physical and mechanical agents	12	0.8	9	0.6
Other and unspecified chemicals	105	6.6	83	5.2
Infectious and parasitic agents	66	4.1	54	3.4
Other agents	24	1.5	23	1.4
Unspecified agents	154	9.7	198	12.4
Poison oak	349	21.9	354	22.2
Ear conditions	34	2.1	33	2.1
Hearing loss, noise	4	0.3	3	0.2
Hearing loss, unspecified agent	3	0.2	2	0.1
Other ear conditions, pressure	19	1.2	19	1.2
Other and unspecified ear conditions	8	0.5	9	0.6

Table II continued on next page

Table II—Continued

Diagnosis and Hazard	Doctor's First Report of Work Injury		Employer's Report of Industrial Injury	
	No.	%*	No.	%*
Effects of weather, exposure and related conditions	59	3.7	52	3.3
Heat	58	3.6	51	3.2
Other agent	1	0.1	1	0.1
Infective and parasitic diseases	19	1.2	20	1.3
Food poisoning	1	0.1	1	0.1
Infectious and serum hepatitis	1	0.1	1	0.1
Dysentery, gastroenteritis, and other infectious disease of digestive system	4	0.3	3	0.2
Other infective and parasitic diseases	13	0.8	15	0.9
Other and unspecified conditions classified as occupational disease	129	8.1	189	11.9
Effects of smoke exposure	17	1.1	17	1.1
Effects of chlorine gas exposure	8	0.5	2	0.1
Diseases of the nervous system other than neuritis	3	0.2	1	0.1
Cardiovascular and other diseases of the circulatory system	12	0.8	12	0.8
Psychoneurotic disorders	2	0.1	1	0.1
Allergic reactions other than skin or respiratory only	5	0.3	3	0.2
Other occupational diseases	28	1.8	23	1.4
Symptoms classified to occupational disease	54	3.4	130	8.2
Conditions not classified as occupational disease by doctor or employer†	75	4.7	58	3.6
Chemical burn	16	1.0	24	1.5
Specified not industrial	46	2.9	4	0.3
Other condition	13	0.8	8	0.5
Unspecified condition	....	....	4	0.3
Report of exposure with no mention of illness	....	....	18	1.1

*Percents are rounded independently and may not add to totals.
†The matching employer or doctor report showed case as occupational disease.

reported by doctors, employers, or both as occurring during October, 1958 (Table I). In the matching process, both doctor and employer reports were located for 1,592, or more than two-thirds of the cases. The proportion of matched reports was higher (three-quarters) when disabling cases alone were considered. Doctor reports were missing for 14% of all cases and for 12% of the disabling cases. Thus, for matched and unmatched cases combined, doctor reporting was found to be about 86% complete. For disabling cases, doctor reporting was 88% complete.

Employers reported about the same proportion of all disabling occupational disease cases as physicians. For nondisabling cases, employers were less likely to file reports than doctors.

It was obviously not possible to consider cases that might have been diagnosed as occupational diseases but for which no reports were received from either doctor or employer. For instance, it is known that in California certain occupational disease categories are underreported. These include silicosis, tuberculosis, hearing loss due to noise, and other conditions where there is a long and indefinite period between occupational exposure and diagnosis of the illness. For various practical reasons,

Table III—Extent of Agreement Between Doctor and Employer Reports of Occupational Disease by Nature of Condition, California, October, 1958

Nature of Condition Reported by Doctor	Total	Nature of Condition Reported by Employer				% Agreement with Doctor
		Same Condition as Reported by Doctor	Skin Condition	Other	Not Occupational Disease	
Total	1,592	1,338	76	120	58	84.0
Systemic effects of poisons	75	45	....	25	5	60.0
Respiratory conditions	79	64	....	8	7	81.0
Skin conditions other than poison oak	773	695	15*	31	32	89.9
Poison oak	349	338	9†	1	1	96.8
Ear conditions	34	33	1	....	....	97.1
Heat exhaustion, other effects of weather and exposure	59	48	....	9	2	81.4
Infective and parasitic diseases	19	15	3	1	....	78.9
Other and unspecified conditions classified as occupational disease	129	100	3	15	11	77.5
Conditions not classified as occupational disease	75	....	45	30	....	0.0

†Reported by employer as skin condition other than poison oak
*Reported by employer as poison oak

the doctor's first report does not lend itself satisfactorily to the reporting of such conditions. For instance, the worker may no longer be employed; his condition may not be diagnosed as an occupational disease until some years later; or the plant may have gone out of business. The extent of underreporting of such disease is difficult to determine but is undoubtedly significant.

Table IV—Disability as Reported by Doctors and Employers in Reports of Occupational Disease, California, October, 1958

1 or More Days Lost from Work	Doctor's First Report		Employer's Report	
	No.	%	No.	%
Total cases reported by both doctor and employer	1,592	100.0	1,592	100.0
Yes	454	28.5	347	21.8
No	922	57.9	1,080	67.8
Not Reported	216	13.6	165	10.4

In order to learn how many doctors are involved in the reporting system, a card file was made giving the name and address of each doctor or group of doctors reporting one or more occupational disease cases during a six-month period (October, 1958, and January through May, 1959). The count showed that 3,214 different doctors or groups of doctors reported one or more cases of occupational disease. On this basis, it was estimated that in a one-year period a total of at least 4,500 doctors report cases of occupational disease and of these approximately 4,000 are doctors of medicine. This is approximately one-fourth of the doctors of medicine in private practice in the state. Many of those not reporting are pediatricians, obstetricians, or doctors in other fields of medicine not likely to treat cases of occupational diesease.

The professional degree held by the reporting doctor was tabulated for a two-month period. Doctors of medicine comprised 88% of the total of those reporting, doctors of osteopathy 9%, and doctors of chiropractic 3%. This is almost the same percent distribution of doctors caring for patients in the general population as was found in the 1954-1955 California Health Survey.

Comparison of Information Reported by Doctors and Employers

Matched reports were used for comparing information provided by doctors and employers. The items compared were diagnosis and cause of illness and disability.

Early in the matching process, it was noted that the employer frequently used exactly the same terminology as the doctor, suggesting that he had received a copy of the doctor's report or was otherwise aware of the medical findings. It was therefore expected that there would be substantial agreement on the diagnosis and hazard on the doctor and employer reports. This observation was corroborated by the study findings.

Table II presents in detail the diagnosis and hazard as reported by the doctor and employer in the 1,592 matched reports. It also points to the wide variety of occupational diseases reported as occurring during a single month in California.

Differences for individual categories were, on the whole, relatively small, although there was a tendency for doctors to give more explicit information than employers. Largest important differences occurred for systemic effects and skin conditions attributed to agricultural chemicals. The difference was also large for the subgroup "symptoms classified to occupational diseases" with more employer than doctor reports falling into this category.

Table III present the results on the extent of agreement between doctor and employer reports for broad diagnostic groups. Both the doctor and the employer gave essentially the same information on diagnosis for 84% of the 1,592 matched reports. The least agreement (60%) appeared for systemic effects of poisons. It is believed that the extent of agreement would have been higher had coding rules established by the California Department of Public Health been less restrictive. For instance, illness was coded to "systemic effects of poisons" only if the report specified "poisoning" or specifically listed symptoms affecting more than one body system.

With respect to the reporting of disability, a comparison of the matched reports showed that doctors estimate more cases as disabling than employers report. The Department of Industrial Relations defines a "disabling work injury" as one which causes absence from work beyond the day of injury. One reason for the difference between doctor and employer reports may be due to the manner in which the question on disability is asked of them. The doctor is asked to estimate the probable disability at the time he first sees the patient. As the term "disability" is not defined on the doctor's reporting form, some confusion may exist regarding part-day absence from work. Moreover, although he makes a recommendation, the doctor usually has no direct knowledge of whether the

employee follows his advice about returning to work.

The employer, on the other hand, is asked to report whether the injury actually resulted in disability beyond the day of accident and to give the dates the person last worked and when he returned to work. Employer reporting of disability is therefore generally more accurate.

The summary of replies to the question on disability for the 1,592 matched reports appears in Table IV. Twenty-eight per cent of the doctor reports estimated there would be disability. In contrast, about 22% of the employer reports indicated time lost from work. Not shown in the table is the fact that among the 454 cases considered disabling by doctors, employers reported no time lost from work for 182, or 40%.

Conclusions

The findings of the evaluation study indicate that the doctor's first report of occupational disease usually contains all the information he has on the case. He does not gain much further information upon revisits by the patient, although more than two-thirds of the patients do return. Aside from the 6% of the cases later judged to be not work-connected, little additional information on the circumstances of the case was obtained on follow-up.

Prior to the evaluation study, no objective measure was available as to either the accuracy or the completeness of doctor reports of occupational diseases. It can now be concluded that within the limitations of the study no large areas of inaccuracy or incompleteness were revealed. Statistics of occupational diseases in California, based on the doctor's first report of work injury, can be regarded as reasonably valid and the best available. Insofar as occupational disease cases are recognized and reported, it can also be assumed that doctor first reports provide worth-while clues to potential health problems in industry for use in programs of prevention and control of occupational diseases.

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NATIONAL TUBERCULOSIS ASSOCIATION FELLOWSHIPS

As the medical section of the National Tuberculosis Association, the American Thoracic Society (formerly the American Trudeau Society) provides fellowships and traineeships for the graduate education of investigators and teachers in the field of respiratory diseases and tuberculosis. The following types are offered:

Edward Livingston Trudeau Fellowships: A few fellowships at a higher level of training and award are offered to specially qualified candidates with an M.D. degree who have been assured of a continued teaching or research appointment upon completion of training. Trudeau Fellowships may be renewed up to a total period of four years.

Research Fellowships: Postdoctoral fellowships are offered to candidates holding the degree of M.D., Ph.D., or Sc.D. for further training as scientific investigators. Predoctoral fellowships are offered to graduate students with a bachelor's or master's degree to work on a research project for an advanced degree other than M.D.

Teaching Traineeships: Physicians entering their second or third year of residency in Internal Medicine or Thoracic Surgery at a medical center and who are interested in a career of medical teaching are offered traineeships toward that end.

Each applicant for an NTA fellowship or traineeship must have been accepted by the head of the department under whom he expects to work for the next academic year in this country. Awards are determined by individual circumstances and are paid directly to the fellow on a quarterly basis. They are granted for one year, beginning on any date after April first, and are renewable for a total of three years.

All applications must be submitted by November 1st. Further information about fellowships may be obtained upon request from: Director of Medical Education, American Thoracic Society, 1790 Broadway, New York 19, New York.

Practical Neurosurgical Principles in the Management of Multiple Injuries

Theodore Kurze, M.D., Los Angeles, Calif.

THE SCOPE and infinite number of variations involved in the neurosurgical approach to problems of combined injury do not permit a dogmatic or complete discussion. If certain fundamental concepts are understood and applied, patient care can be greatly facilitated, regardless of the complexity of the problem and the number of clinicians participating.

Clinical Shock and Closed Head Injury

The role of shock in head injury is far from settled in the medical literature, as evidenced by the divergent views expressed in current and not-so-current textbooks of surgery.[4, 3, 6, 5, 13] If we accept the signs of clinical shock as a manifestation of reduced circulating blood volume resulting from an enlargement of the vascular system or a reduction of circulating blood in the compartment, we might then ask how this can occur in a closed head injury.

Could there be a reflex vasodilatation evoked by the stimulus of cerebral trauma? More than half a century ago, Polis[10] discovered that a concussive blow to an animal's head resulted in a rapid rise in blood pressure. These findings were confirmed and amplified by Denny-Brown and Russell[7] and Walker, Kollross, and Case.[14]

Campbell and Whitfield[2] found clinical evidence of shock in the first six hours following injury in 46 of 718 cases of cerebral trauma (6%) and concluded that shock is neither an essential nor a prominent feature of closed cerebral injury. It is therefore reasonable to conclude that cerebral trauma of sufficient magnitude to produce concussion or contusion usually causes a rise in blood pressure.

Smolik, Brown, and Nash[11] noted a drop in red blood cells, hematocrit, and hemoglobin levels in cases of isolated closed cerebral injury. In the 68 cases studied, 40 (58.8%) showed this change without evidence of hypovolemia or known blood loss. Further study[12] of blood volume in 21 similar cases with radioactive iodinated human serum albumin and red blood cells tagged with an isotope of chromium revealed an expansion of circulating blood volume in 17 patients. The increase occurred in the first five days and in patients with severe injuries.

Is it possible for intracranial bleeding to be of sufficient magnitude to cause hypovolemic shock? In the average adult the intracranial blood volume is 1,500 cc. A minimum loss of 500 to 1,000 cc of circulating blood is required to reduce the hemoglobin 1 gm to produce the very earliest signs of clinical hypovolemia. It is

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quite obvious that the brain would be completely destroyed or that the skull would explode before sufficient intracranial bleeding could occur to produce the very earliest signs of blood loss in a closed head injury.

Therefore it is not safe to ascribe true surgical or hypovolemic shock to head injury alone. The development of clinical symptoms of hypovolemia should immediately initiate an intensive search for a ruptured hollow or solid organ in the thorax or the peritoneal or retroperitoneal space.

Clinical Observations for Evaluation of Cerebral Injury

Today there is no established specific therapy for the uncomplicated cerebral injury, other than general supportive care directed toward maintaining physiologic homeostasis, although there is good reason to suspect that there soon may be a more definite therapeutic approach directed at cerebral resuscitation. The purpose of the clinical evaluation is, therefore, directed toward the prompt recognition of complications and establishing the indications for surgical intervention.

Except in unusual circumstances, the initial assessment of the patient with a head injury is the responsibility of a physician not trained or regularly practicing one of the neurological disciplines. The physician who does not have occasion to perform a detailed neurological examination as part of his daily routine may experience a sensation of profound despair as he attempts to mobilize the eponyms lurking in the remote corners of his parieto-occipital lobes. This is, indeed, unfortunate for both physician and patient, because the complex, detailed neurological examination is unnecessary in the proper evaluation of a patient with a recent head injury. All the clinical observations necessary for proper neurosurgical judgment and management can be obtained by recording a few simple obvious facts about the patient's condition. This is meant not to suggest that the surgical judgment and decisions made are easy to come by but to point out that the necessary observations are easy to gather and record.

I. State of consciousness
  A. Awareness of orientation
    1. Time
    2. Place
    3. Person
    4. Purpose
  B. Response to external stimuli
    1. Verbal
    2. Tactile and pain
    3. Cough
    4. Corneal
    5. Light (pupils)
  C. Response to internal stimuli
    1. Restless
    2. Delirious
    3. Lethargic
    4. Garrulous
    5. Confabulative
II. Motor examination
  A. Pupils
  B. Facial symmetry
  C. Extremities
  D. Deep tendon reflexes
  E. Plantar response (Babinski)
III. Systemic signs
  A. Blood pressure
  B. Pulse
  C. Respirations
IV. Laboratory and x-ray
  A. Electroencephalogram
  B. Lumbar puncture
  C. X-ray examination of the skull

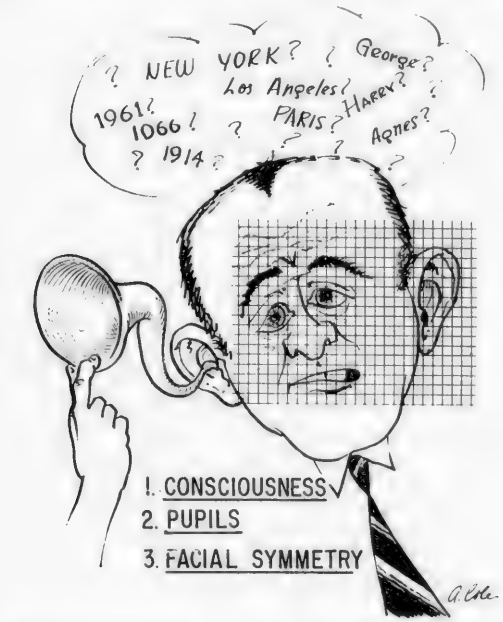
The importance of the laboratory and x-ray observations, in special instances, is not to be minimized. In the initial evaluation, however, they usually do not contribute data of sufficient importance to alter the neurosurgical management.

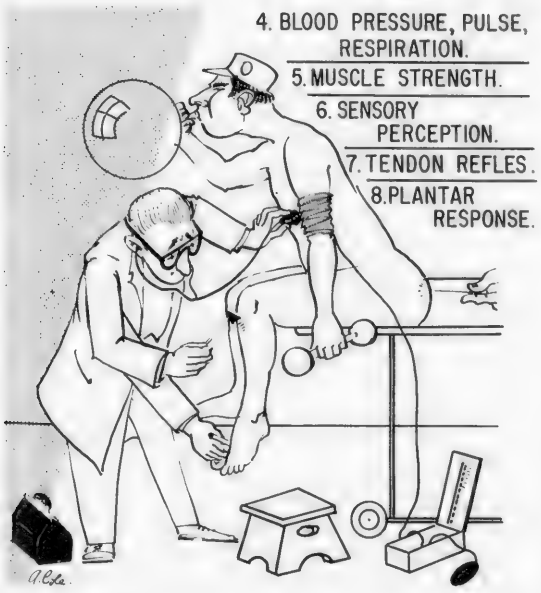
Complications of Closed Head Injury

As indicated earlier, the primary neurosurgical responsibility is promptly to recognize and treat the central nervous system complications of closed head injury. The post-traumatic intracranial hemorrhage—that is, the extradural, subdural, and intracerebral hemorrhage—are

the problems which demand recognition and treatment, particularly during the first 72 hours following the injury. Although the pathogenesis, clinical features, and surgical therapy are obviously not identical, they are sufficiently similar to be grouped for the purpose of general discussion, particularly from the standpoint of early recognition.

In general, it may be said that whenever a patient with a closed head injury exhibits signs of increasing intracranial pressure or progressive neurological impairment one of the above complications must be strongly suspected. Referring to the previously suggested outline, any or all of the following clinical changes may be encountered.





I. State of consciousness, impaired
II. Motor examination
  A. Dilation and/or failure of one or both pupils to react to light
  B. Development of facial asymmetry, that is, particularly of the mouth upon response to pain
  C. Weakness or paralysis of the extremities (The technique of demonstration is commensurate with the patient's state of consciousness.)
  D. Consistent inequality of the tendon reflexes, particularly between right and left
  E. Abnormal plantar response
III. Systemic signs
  A. Rise in blood pressure or pulse pressure
  B. Bradycardia
  C. Reduced respiratory volume and rate
IV. Laboratory and x-rays
  A. The electroencephalogram is seldom of value in the assessment of the acute head injury because of the rapidly changing electrophysiology in response to injury and the frequency of artifacts of somatic origin, although it may be extremely useful in recognition of a chronic subdural hematoma.
  B. Lumbar puncture is of limited value in that in almost all cases of cerebral contusion some blood is demonstrable in the spinal fluid, with elevated pressure, whether there is or is not a post-traumatic intracranial hemorrhage. It is of particular value in problems of coma of unknown etiology where there is circumstantial evidence to suggest cerebral injury.
  C. The presence or absence of a linear fracture seldom influences the surgeon as to whether there is or is not a post-traumatic hemorrhage, although it is certainly well recognized that a fracture across the middle meningeal groove in the temporal fossa frequently accompanies an extradural hemorrhage; but the presence of a fracture or the absence of a fracture would not, of itself, influence the decision toward surgical intervention.

There is great variation in the severity and the number of clinical changes which may indicate the existence of post-traumatic interval hemorrhage; and, in most instances, the clinical diagnosis cannot be absolutely established short of the placement of diagnostic and/or therapeutic burr holes. Cerebral edema alone may present any combination of the above positive clinical signs, or it may coexist with post-traumatic hemorrhage and increase the perplexity of the problem.

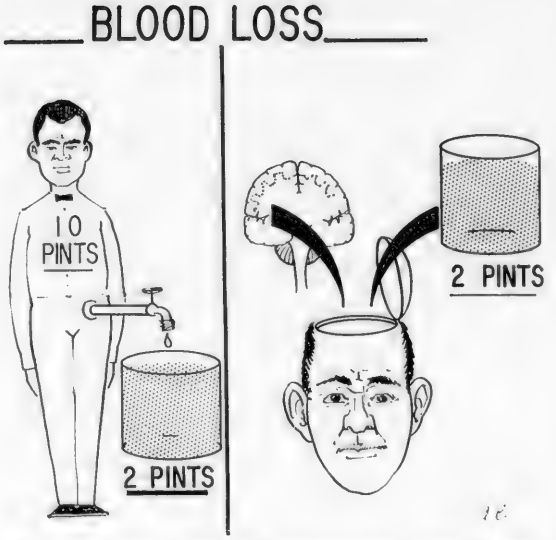
It is, however, vital to distinguish the above complications of head injury from the systemic complications which may accompany head injuries and contribute to the over-all deterioration of the patient's condition.

The following compartment and system review is an effort to outline some of the more significant complications that have been encountered in association with head injury.

Pulmonary Complications—Chest injuries are frequently seen with cerebral injury. A chest injury of sufficient magnitude to interfere with cardiac and pulmonary function often tends to increase the severity of the cerebral symptoms. This can be accounted for by the following factors: (1) the great dependence of the brain on the heart and lungs to supply the oxygen and other ingredients necessary to support its relatively high metabolic demands, and (2) interdependence of the pressure that exists between the thoracic and cranial cavities.* Therefore the immediate treatment of pneumothorax and/or hemothorax or scaphoid chest with paradoxical respirations not only may be essential to save the patient's life but will also reduce the severity of permanent cerebral damage resulting from the total injury.

Patients with severe cerebral injury should be considered as already having, or excellent candidates for, accumulation bronchopulmonary disease or pneumostasis.* The relationship becomes obvious if the causes of accumulation presented by Moore[9] are projected to the clinical setting of the neurosurgical patient. The frequency of aspiration pneumonitis, diminished cough reflex, diminished respiratory excursion, the sticky and tenacious respiratory secretion of dehydration, and the intermittent obstruction of the trachea by the retrolapsing tongue are all too familiar to those dealing with severe cerebral injury. Therefore whenever a patient with a severe head injury develops tachypnea, tachycardia, and/or fever, accumulative pneumostasis should be considered. The diagnosis can often be established by identification of segmental reduction of breath sounds, lateralized lag in respiratory excursion, bronchial breathing, and cyanosis unrelieved by oxygen. It should be remembered, however, that under certain circumstances oxygen therapy may be dangerous. In the absence of these clinical findings, the diagnosis should not be abandoned without roentgenograms of the chest wherever possible. Unrecognized accumulation pneumostasis may lead to acute respiratory acidosis with depression

*According to Moore[9] (p. 325), this term was proposed by Walter J. Crandel, M.D.



of the sensorium and secondary neurological changes, which can easily be confused with increasing intracranial pressure.

The ideal therapy is anticipation and recognition. Often, some assurance on the part of the neurosurgeon to the other members of the clinical team is necessary to initiate increased bed activity, coughing, tracheal aspiration, and tracheostomy before recognizable pneumostasis has occurred.

In the presence of coma, respiratory depression, varying degrees of aspiration pneumonitis, reduced cough reflex, and reduced pharyngeal reflexes, early tracheotomy is essential for proper management.

Pulmonary embolism is not uncommon in neurosurgical patients. The usual source is from the veins of the legs or pelvis. The prolonged immobility (often unnecessary) and frequent (also unnecessary) use of veins of the lower extremity for parenteral fluids may be contributing factors to the frequency with which this problem is observed in neurosurgical patients. The symptoms vary from sudden, extreme dyspnea, shock, retrosternal oppression, and quick death to the subtler clinical manifestations of severe leukocytosis, pleurisy, and bloody or rust-stained sputum.[1] The existence of this problem in a patient with a serious cerebral injury in its acute phase is, fortunately, uncommon. When it does occur, however, it represents one of the most challenging problems in the selection of therapy and management that may confront the people responsible for the management of combined injury; and the decision as to whether to use anticoagulants and/or prophylactic ligation must be thought through very carefully by all members

of the team, regardless of their disciplines.

Metabolic Complications—Acute hypokalemic alkalosis is most frequently encountered as a complication of traumatic and surgical upper gastrointestinal syndromes. It is, however, occasionally encountered in the neurosurgical patient; and its recognition is mandatory. The clinical picture of weakness, apathy, coma, tetany, and convulsions could easily be confused with increasing intracranial pressure.

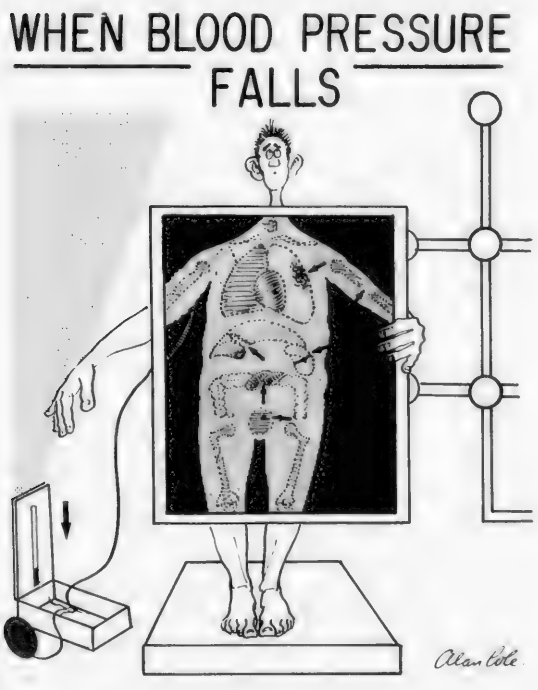
The pathogenesis of hypokalemic alkalosis can be readily related to the internal environment accompanying head injury.

In the first place, emesis, which is often considerable in the early stage of severe head injury, produces an initial loss of acid that ordinarily can be compensated for by an increase in renal output of sodium bicarbonate. The aldosterone-like effect associated with trauma, however, reduces renal excretion of sodium bicarbonate and accelerates the alkalosis. This favors an increase in urinary potassium output, which is already accelerated by the trauma.

The potassium loss further reduces the ability of the kidney to secrete an alkaline urine, and the stage is set for a self-accelerating cycle.[9]

The characteristic plasma pattern indicating this phenomenon is a high pH and CO_2-combining power (7.53 and 32 mEq.), low chloride and potassium (85 and 2.0 mEq. or lower).[9]

Administration of potassium chloride will restore potassium loss and enable the kidney to secrete base. This usually interrupts the cycle and points the patient's return toward homeostasis.



Most students of the metabolic care of neurosurgical patients no longer adhere to the regimen of dehydration and sodium restriction. Parenteral fluids, when required, should be programed to maintain physiologic homeostasis. In patients with cerebral injury, fluid and electrolyte requirements are not unique. The needs of the total patient should be considered.

Intraabdominal Complications — The intraabdominal complications of cerebral injury may be classified as follows: (1) complications arising from an alteration in central regulation of the gastrointestinal tract, and (2) complications arising from concurrent injury.

1. Acute gastric dilatation or atony is one of the serious occasional complications of cerebral injury. Unrelieved, it may be lethal in an hour or two. Often the vomiting is effortless—the vomitus simply pours out of the mouth without retching. The material is foul and dark and contains altered blood. Tachycardia and prostration often accompany it. Thirst may be insatiable. The epigastrium bulges, and tympanites extends far up into the left thoracic cavity. The treatment is immediate aspiration of the stomach followed by continuous gastric suction for 24 to 48 hours and restoration of fluid balance.

Injury to the paired nuclei of the hypothalamus at the base of the skull can cause an acute increase in peptic activity and gastric peristalsis, which may result in a localized, acute peptic ulcer or generalized gastromalacia. This may, and often does, lead to massive gastrointestinal hemorrhage and perforation.

2. Perforated bowel, ruptured spleen or liver, and traumatic pancreatitis represent a portion of the spectrum of intraabdominal injury which may accompany cerebral injury. Each of these has its own clinical signs and relative indices of suspicion; and it is not the purpose of this paper to discuss them. The alteration in the patient's conscious state, however, may make recognition of these most serious problems very difficult.

The same general principles are applicable to traumatic lesions of kidney, ureter, and bladder in the retroperitoneal compartment, gross or microscopic hematuria being the principal alerting signal.

Extremities—A review of 22 autopsy-proved cases of fatal postoperative pulmonary embolism from a series of 943 consecutive autopsies on adults revealed that 9 (41%) of these oc-

curred in neurosurgical patients.[15] Further study of the same authors revealed that death from pulmonary embolism was seen to occur in neurosurgical patients with about the same frequency as noted for the other major surgical procedures. Pulmonary embolus, however, was a cause of death in 30% of the postoperative neurosurgical deaths and, therefore, represents a complication of major importance. Inasmuch as thrombophlebitis and phlebothrombosis of the lower extremities represent a major source of pulmonary emboli, meticulous attention to prevention is more than justified.

An infrequent but related source of confusion is fat embolism, always to be remembered when extremity fractures are present.

Summary

1. Clinical shock is neither an essential nor a prominent feature of closed cerebral injury.

2. When shock occurs in patients with head injury, an intense search should be initiated for an extracranial cause.

3. The clinical observations necessary to evaluate acute cerebral injury are not complex.

4. Patients with head injuries, regardless of magnitude, can, and frequently do, develop complications in all physiologic systems and anatomical compartments. The secondary effects of cerebral injury often obscure associated injury. This imposes an extra vigilance and awareness on all members of the clinical team.

5. The metabolic care of the neurosurgical patient is not unique, and the total needs of the patient should be considered.

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Mental Health in Industrial Practice

III. THE PART-TIME PSYCHIATRIST IN INDUSTRY

James A. Knight, M.D., and V. C. Baird, M.D., Houston, Texas

THERE is an important place for the psychiatrist in industry. This view is generally accepted by a broad segment of the medical profession interested in occupational health. And even though there may be fewer than a dozen full-time psychiatrists in industry, as we often hear, we propose that there are many advantages to the part-time use of a psychiatrist.

The psychiatrist may be employed in any one of several working relationships. He may work as a part-time member of the medical staff, devoting only a few hours each week to the company. It may be practical to place him on a regular monthly retainer basis or to have a consultation arrangement. The important thing is to have a regular procedure, so that the psychiatrist may become familiar with company policy, company benefit programs, and the general philosophy of the organization. He will be stimulated by the challenges he finds in industry, and, in turn, he will bring to industry the stimulation of his regular practice. Thus, both he and industry profit from the relationship.

For the past five years, Humble Oil & Refining Company has used a part-time psychiatrist, customarily one from the faculty of Baylor University College of Medicine. We would like to present our attitudes and findings regarding the value of such a consultant.

The Psychiatrist as Physician

The psychiatrist has his office in the medical department, works through this department, and is directly responsible to the medical director. We are aware that this is not the case in all industries. But we feel that, since the psychiatrist is a physician and his specialty is a part of medicine, he ought to be accepted first as a physician.

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The mind and the body are one, and they function as interactive and interdependent organs. Plato's words to the physicians of his day are as pertinent now as they were centuries ago: ". . . This is the great error of our day in the treatment of the human body, that physicians separate the soul from the body."

Emotional illness is illness of the person, not of the mind alone. So whenever an effort is made to modify behavior patterns in a disturbed person, we approach the therapy as a physician with total medical responsibility and an appreciation of the necessity for complete confidence in the doctor-patient relationship.

Each person with a problem is treated as an individual and his situation dealt with according to his unique needs. We avoid screening procedures, impersonal testing, and generalizations about problem areas and problem people.

George Preston,[1] in his treatise on "Psychiatry for the Curious," writes:

> We must always study one particular individual in one particular place at one particular time acting as the result of one particular series of causative factors. You could not draw pictures of "delinquent children" but you could draw a picture of a boy stealing a watermelon out of a field in the summertime. You might be able to generalize a little about boys who stole watermelons. Even if you did this, you would know nothing about the particular boy who stole the particular watermelon from the particular field. Human behavior, good, bad, indifferent, normal, insane (whatever those terms mean), is never carried out by boys, girls, or men, or mankind, but always by a Particular Boy, Girl, Man or Woman in a certain place at some definite time and for a reason that is peculiar to that one, and only that one, person. You may make averages for a hundred or a thousand individuals, but one of the big dangers of psychiatry is to believe that you can treat any particular human being on the basis of averages.

This is a simple statement, but it summarizes our basic philosophy that each person and each situation must be approached individually and uniquely. Actually, this is the point of view

of Humble's entire medical staff in its over-all approach to occupational health.

Three Types of Referrals

Referrals to the part-time psychiatrist from full-time company physicians fall into three groups.

The first type is the employee who must be seen and evaluated psychiatrically because of an obvious mental illness, a profound disturbance in his interpersonal relationships, or a serious change in his work pattern. Such a person is told that a psychiatric evaluation is necessary.

A cooperative effort between the full-time physician and the consulting psychiatrist is seen in the handling of the following case in this category.

A 41-year-old engineer developed many grandiose ideas about what he could do for his company. He threatened to quit his job and move to Miami with his family in order to write a book about morale among his fellow employees. He was tense, sleepless, but in bright spirits. He was filled with all kinds of schemes about the many wonderful things that he wanted to do for understanding among men and improvement of his company. It seems that the precipitating factor in his illness was a big engineering conference for which each department had to prepare extensive reports. This man was in charge of part of the program, and he did an excellent job but worked himself almost to exhaustion. After the conference, his manic behavior became disturbing to family, friends, and associates, and they suspected that something was wrong.

The psychiatric examination revealed no disorder in thinking but a very pronounced disorder in effect. It was obvious that this patient was in the manic phase of a manic-depressive psychosis. Yet the patient was collected enough to talk with us and to enter into some of the planning for his treatment. We discussed with him our interpretation of his illness and how he ought to let us advise the course of action. We told him that he definitely needed treatment, and he was willing to accept this without much reluctance. Plans were made for hospitalization, and he was referred to a psychiatrist in private practice.

In the past, he had not had any particular depressive episodes. Even though his behavior had been in part cyclothymic, he generally had been a hypomanic personality. He was very intelligent and a likable person. He responded to psychiatric treatment and made an uneventful recovery.

The value of the psychiatrist in this case was his relationship with the company. This engineer promised his friends and associates that he would seek and accept only the guidance of the headquarters medical department, which he held in high regard. The availability of an adequate staff in the medical department thus was significant in the expeditious handling of this patient's serious illness. A thorough and persistent interpretation of his illness and its meaning in the light of his personal goals for himself and the company was done in a way that we felt would be acceptable to him. His confidence then permitted us to prescribe the treatment regimen which would aid in recovery and rehabilitation without delay.

The second group of referrals from full-time company physicians is made up of persons who may have an organic illness with marked emotional overlay, a functional illness, or some kind of psychosomatic problem. The physician who is consulted tells this employee that a psychiatrist is available in the medical department. He then suggests that seeing the psychiatrist may be helpful in the management of the particular illness involved. The employee may accept the opportunity or decline it. The following case is representative of this category.

A tall, handsome 39-year-old worker was referred because of tension headaches. His excellent command of language was impressive. Though his formal education extended only through high school, his store of knowledge was great. He had many interesting hobbies, from organ building to collecting 19th century Springfield rifles. He was well informed in electronics and mathematics. The most impressive aspect of his life was that, though he had great ability and a sizable store of information, he was holding a mediocre job in the plant—working in a toolroom. His assigned duties were not a challenge and probably did not require even a small portion of his skills. A review of his history revealed that on several occasions in different settings he had worked himself up to a promotion of a fairly high level and then had quit and sought another job. Frequently there were realistic factors in his quitting and moving, and at other times the factors did not seem realistic. This was the story of a man who sabotaged himself at critical periods in his advancement.

What does it mean when one practices this type of self-sabotage? Intensive and deep probing is often necessary to uncover such dynamics. Some of the commoner factors are fear of success because of what it means—such as leaving one's peer group through advancement and then running the risk of being rejected by fellow employees; fear of competition, which always exists when one moves into a more favorable work status; fear of increased responsibility and the greater possibility of failure; an unconscious desire to fail as a form of punishment for some real or fantasied wrong committed in the past; fear of overcoming and advancing beyond the father (this may sound strange, but it is fairly well documented by psychoanalysis that certain per-

sons seem to hold themselves back so as not to surpass or at times equal the father's accomplishments though their potential is greater); deep feelings of inadequacy which come to the surface when a bigger job is offered—"This new job is a man's job and I am still a boy"; and a desire to fail as punishment of one's family toward whom he is hostile, for they use him only as "a work horse."

We have found it helpful to point out some of the dynamic factors often seen in self-sabotage, without direct application to the person with whom we are talking. This initiates a process of introspection aided somewhat by the brief psychotherapy, which not infrequently uncovers some of the specific dynamics related to this person's failure. In the case of the patient described here, we could not identify significant psychodynamic factors, but the impact of the insight that he was probably unconsciously sabotaging his own advancement became a powerful stimulus for change. Without any intervention on the part of anybody in the medical department with his supervisors, he assessed his working environment, began applying himself in a more creative and intelligent way, and in a matter of a few months earned for himself a major promotion to a job which challenged his ability and skills. As his work situation improved, his tension headaches diminished.

The third type of patient is the kind who consults the psychiatrist on his own initiative and without referral for a variety of problems involving himself or his family. Such an employee could arrange for the appointment through the receptionist in the medical department, but he generally mentions to one of the full-time physicians his interest in seeing the psychiatrist, and arrangements are made.

Often the problem he brings concerns a member of his family. He may need counsel about commitment proceedings or about the resources available for handling a particular type of family crisis or about a personal problem involving himself alone or other members of his family. In other words, those who seek an interview on their own often have problems similar to those who are referred. The following patient's concern is typical of this group.

A secretary came in to discuss her parents and sister. She was not living at home but was acutely aware of what was going on there. The description of her father rather classically fitted the picture of a paranoid personality. He was suspicious and jealous and frequently accused his wife of infidelity. There was always conflict in the family, and the principal victim was the adolescent sister. She was trapped in the crossfire between the parents and began to develop numerous physical and emotional symptoms. Her situation illustrated the old maxim that "what the parents do to one another is reflected in the child." It was obvious that the whole family needed psychiatric help, and, since this employee had a fairly good relationship with every member of the family, she seemed to be the logical one to persuade them to seek such help.

We usually give such a person some of the dynamics of the syndrome being dealt with—in this case, the paranoid character (represented by profound concern over masculinity and a conspicuous tendency to utilize a projection mechanism, expressed by suspiciousness, envy, extreme jealousy, and stubbornness). This person is also told that often there is some psychopathology in one or more other members of the family, and often one member irritates another or acts out through that member. All of this combined makes it essential that the whole family and its interaction be evaluated psychiatrically.

Since there is often a delay before psychiatric help is obtained, such an employee who is relatively healthy and stable can act as therapist in some ways. First, he can prevent himself from being caught up in the conflict. Then he can treat the obviously disturbed one as a patient. (It is amazing how one's expectations and attitudes become more reasonable and supportive when one looks upon another as a patient.)

This employee was able to get psychiatric help for her family. A part of the treatment plan was a temporary separation to permit some of the wounds to heal. Communication channels were kept open and in excellent repair. The family is now preparing to come back together, with greater understanding of each other's needs and with considerable harmony.

Consultation between the referring physician and the psychiatrist generally takes place before and after the interview with the patient. Very little psychiatric information is recorded in the medical department records, though the records are kept confidential. Recording a great deal of data may put a patient on guard. The psychiatrist does keep adequate notes in a personal file.

The Psychiatrist as Consultant

The psychiatrist has other responsibilities in addition to those discussed. The medical staff consults him about problems of their patients that he does not see. He is involved in the over-all and continuous training of staff (medical and nonmedical) regarding basic psychiatric principles and practices. He is also avail-

able for broad, general counsel about policies and matters having to do with any area of human relations. An occasional conference may be held involving the physicians and a group of supervisory or management personnel. Following is a report of such a conference and how it came to be held.

We saw in the medical department a number of very able workers from one division of the company. These workers came with psychophysiologic disorders, hypochondriacal complaints, depression, or organic illnesses aggravated by emotional factors. We felt that the work situation had a part to play in the symptoms of these patients.

This type of situation seemed to offer us an excellent opportunity to meet with the leaders in this division and share with them our findings about the relationship of stress to illness, of how individuals with certain personality characteristics often choose a particular kind of work, to obtain from them a true analysis of the working environment and its demands, and to put our heads together to improve the situation for the benefit of both the worker and the company.

The conference was opened by one of the internists in the medical department who discussed, in a general fashion, stress and the recent research in that field. Others discussed the demands and requirements of the jobs in marketing accounting, which consisted of meeting deadlines in processing thousands of invoices, getting out financial reports, and handling a variety of financial matters of the company. The psychiatrist then discussed another major factor—personality. The person attracted to this type of work is often a perfectionist who is somewhat critical of himself and therefore makes heavy demands upon himself, who often has a tendency to be depressed periodically, and who handles many of his internal conflicts by compulsive rituals and obsessive thoughts. A general discussion took place as to how this type of employee brings to his work particular personality characteristics and then finds in his job stressful problems. The two factors combine to lower his efficiency and give him symptoms. The management personnel present mentioned that they could readily give more encouragement, appreciation, and recognition to these workers for their strenuous effort. Perhaps, they suggested, they ought to try to limit the amount of overtime required. Both of these suggestions were profoundly significant and helpful. After the conference, some of those present remained to discuss some other areas of their responsibility. The conference was of value and was received well. Such a conference represents one of the truly worth-while functions which the medical department can perform as a method of interpreting to the management of the company the types of situations which can contribute to poor emotional and physical health.

None of this activity divorces the psychiatrist from the role of physician, for he is interested not just in treatment but in prevention of mental illness. A psychiatrist may help an industry work out and identify these hidden emotional factors which decrease production and create social stresses, dissatisfactions, and ill health. This does not mean he is capable of managing an enterprise, establishing policy, or operating a computer. He is a medical specialist in human behavior and accepts certain problems in industry in the same way as he would accept a nuclear physicist as a patient for psychotherapy. Though the psychiatrist knows very little about physics, he is able to help his patient become a better physicist by helping him discover those emotional factors in himself which inhibit him in his work.

Conclusion

We do not look upon psychiatry as a panacea for all the ills of individuals or of industry. It can, however, assist industry to a better understanding of the development of individual personality, motivation for work, interpersonal relationships, and obscure behavior. Our overall goal is the maintenance of the health of people in industry, and, since we view the person as a whole, it is hardly necessary to preface health with the words "physical" and "mental." We are especially concerned with normalcy and maturity, and our goals of therapy and prevention are oriented in this direction. All specialists in the field of human behavior have their definitions of the normal or the mature person. Sigmund Freud was once asked what it was he would consider the ultimate in normal behavior and he answered, "Arbeiten und lieben"—to work and to love. Thus, *normal* or *healthy* in the Freudian sense has always carried a connotation of human values, such as unremitting creative effort and the love of one's neighbor. This simple, easily understood definition is worthy of our most serious consideration today as we apply our efforts to the industrial scene.

(This concludes this series of articles.)

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Blood Pressure, Body Weight, Serum Cholesterol, and Smoking Habits Among Executives and Nonexecutives

Sidney Pell, Ph.D., and C. A. D'Alonzo, M.D., Wilmington, Del.

IN CONTRAST to popular belief, there has been a growing volume of evidence to indicate that executives, managers, and professional men are no more prone to develop coronary heart disease than are other groups of employed persons.[1-5] In fact, the studies of Mortensen and his co-workers[3] and Lee and Schneider[4] suggest that the prevalence of coronary disease is particularly low among executives. Our own study of duPont Company employees is in agreement with these findings.[5] During a three-year period, the average annual age-adjusted incidence rates of acute myocardial infarction per 1,000 male employees were 2.0 among executive personnel, 2.4 in a group composed predominantly of supervisors and nonsupervisory professional men, 3.9 among lower-level salaried employees and 2.9 among production workers.

If executives are less susceptible to coronary disease, why should this be so? An indirect method of approaching this question is to compare executives and other employed persons with respect to certain characteristics associated with the disease. There is general agreement that the major factors which predispose to coronary atherosclerosis are overweight, hypercholesteremia, hypertension, excessive cigarette smoking, and heredity.[6] These factors are indicators, to various degrees, of dietary habits, metabolic disturbances, a person's psychic state, and the amount of physical exercise in which he indulges. Are these factors more or less common among executives; and if less common, can this account for their lower rate of coronary heart disease? The present study is directed to these questions.

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Submitted for publication Feb. 3, 1961.

Method of Study

The group we have studied consists of 1,585 men aged 40 through 64 years who are employed at the home office of the duPont Company in Wilmington, Del. All of these men are salaried, "white-collar" employees.

Every employee of the duPont Company is given a complete physical examination annually by Company physicians. It is from the records of these examinations that we obtained the stature, body weights, and blood pressure readings of the men in our study. Serum cholesterol determinations were made once on each man, during the year 1959. Smoking histories were obtained by distributing detailed questionnaires. Usable replies were returned by 1,499 (95%) of the 1,585 men in the study. Information on family history of cardiovascular disease is being gathered but is not available as of this writing.

There are 489 men in the group whom we have classified as executives. They have been divided into two categories, consisting of 85 top-level executives and 404 lower-level executives for comparison with the remaining 1,096 nonexecutives. The top-level executives include vice presidents, general managers of the various Company departments, heads of sales organizations, and other men who are charged individually with the responsibility of decision making. The lower-level executives include assistants to the top-level men, managers of large office groups, and high-level staff specialists. They make important contributions to the development of Company policies but do not assume final responsibility for those decisions. The nonexecutives comprise all men below these groups, including clerical, sales, professional, and technical workers with varying degrees of job responsibility up to intermediate levels of supervision.

Findings

Hypertension—The men in the study were classified hypertensive if on any two periodic physical examinations the systolic blood pressure equaled or exceeded 150 mm Hg and the diastolic blood pressure equaled or exceeded 90 mm Hg. The prevalence of hypertension in each occupational category is shown by age in Table I. In both the 40 to 49 and 50 to 64 age groups, differences in prevalence rates are small and within the limits of sampling error ($P > 0.10$).

The three job levels were also compared with respect to the blood pressures taken during the last physical examination that was given prior to the collection of data for this study. Cumulative frequency distributions of systolic blood pressures and diastolic blood pressures (Fig. 1) are almost identical in the three levels. Thus, our data show no association between job responsibility and the frequency of elevated blood pressure.

Overweight—In studying overweight in our subjects, we used measurements of stature and body weight. A man was classified as overweight if, at his latest physical examination, his body weight exceeded his ideal weight by 20% or more. Ideal weight for a given stature was specified as the weight midway in the allowable range for a person of medium frame, as listed in the table of desirable weights for men and women prepared by the Metropolitan Life Insurance Company.[7]

Differences in the prevalence of overweight in the three occupational levels (Table II) are not significant ($P > 0.20$), but there is a suggestion of slight association between job level and frequency of overweight that is positive in the 40 to 49 age group and negative in the 50 to 64 age group. An explanation for these contradictory relationships, if real, is not suggested by our data.

Table I—Prevalence of Hypertension Among Executives and Nonexecutives by Age

Age Group	Job Category	Total	Hypertension No.	Hypertension %
40 to 49	Executives:			
	Top level	23	2	8.7
	Other	188	11	5.9
	All executives	211	13	6.2
	Nonexecutives	616	66	10.7
50 to 64	Executives:			
	Top level	62	10	16.1
	Other	216	39	18.1
	All executives	278	49	17.6
	Nonexecutives	480	98	20.4
Both Age Groups	Executives:			
	Top level	85	12	14.1
	Other	404	50	12.4
	All executives	489	62	12.7
	Nonexecutives	1,096	164	15.0

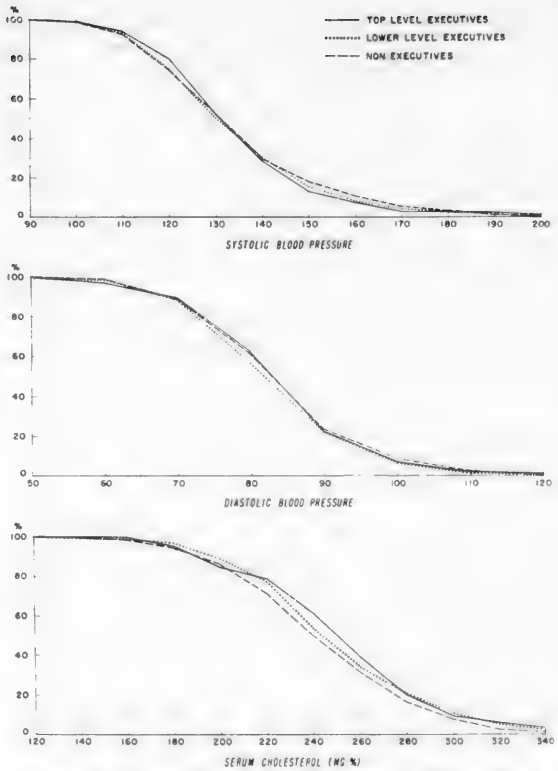

Fig. 1—Cumulative percent frequency distributions of systolic blood pressure, diastolic blood pressure, and serum cholesterol among executives and nonexecutives.

Cholesterol—During the year 1959, the total serum cholesterol was determined for each man in the study. Blood was drawn during the fasting state usually between 8:00 a.m. and 8:30 a.m. The determinations were carried out by the method of Pearson, Stern, and McGavack.[8]

The mean cholesterol levels in each occupation category are seen in Table III. The mean among nonexecutives was 241.8 mg % while the mean levels of the executives were slightly higher: 247.7 mg % in the lower-level group and 250.2 mg % in the top-level group. Analysis of variance reveals that the three means differ significantly ($F = 4.22$, $0.05 > P > 0.01$), but the magnitude of these differences is not of clinical importance and is inconsistent with the observed inverse relationship between job level and incidence of coronary heart disease.

The prevalence of hypercholesteremia (serum cholesterol ≥300 mg %) was found to be somewhat greater among the executives (Table III), but differences among the three groups are within the limits of sampling error ($P > 0.20$) (Fig. 1).

Table II—Prevalence of Overweight Among Executives and Nonexecutives by Age

Age Group	Job Category	Total	Overweight No.	Overweight %
40 to 49	Executives:			
	Top level	23	2	8.7
	Other	188	27	14.4
	All executives	211	29	13.7
	Nonexecutives	612	113	18.5
50 to 64	Executives:			
	Top level	61	14	23.0
	Other	215	44	20.5
	All executives	276	58	21.0
	Nonexecutives	480	80	16.7
Both Age Groups	Executives:			
	Top level	84	16	19.0
	Other	403	71	17.6
	All executives	487	87	17.9
	Nonexecutives	1,092	193	17.7

Table III—Mean Serum Cholesterol and Prevalence of Hypercholesteremia Among Executives and Nonexecutives by Age

Age Group	Job Category	No. of Persons	Serum cholesterol (mg %) Mean	Serum cholesterol (mg %) S.D.	% Hypercholesteremic*
40 to 49	Executives:				
	Top level	23	255.4	50.0	8.7
	Other	188	244.7	43.2	9.6
	All executives	211	245.9	44.0	9.5
	Nonexecutives	616	238.7	40.8	7.0
50 to 64	Executives:				
	Top level	62	248.2	40.4	9.7
	Other	216	250.2	41.0	11.6
	All executives	278	249.8	40.8	11.2
	Nonexecutives	480	245.8	38.1	8.8
Both Age Groups	Executives:				
	Top level	85	250.2	43.2	9.4
	Other	404	247.7	42.1	10.6
	All executives	489	248.1	42.2	10.4
	Nonexecutives	1,096	241.8	39.8	7.8

*Serum cholesterol 300 mg % or more.

Smoking Habits—Our questionnaire on the use of tobacco elicited information on the types of smoking done during the person's lifetime: the daily frequency of smoking, both in the past and at the present time, and the number of years each kind of tobacco was used. The major findings are summarized in Tables IV, V, and VI.

Apart from two noteworthy exceptions, the smoking patterns found in each occupational level were quite similar. There were no significant differences in the choice of types of tobacco or in the proportions of smokers and nonsmokers in each group. There were, however, proportionately more heavy cigarette smokers (two or more packs per day) among lower-level executives (6.8%) than among top-level executives (3.7%) and nonexecutives (3.2%). These differences among the three groups are significant ($P < 0.01$).

Another difference among the three occupational categories is found in changes in smoking habits (Table VI). The proportion of top-level executives who have given up the use of cigarettes is significantly higher than the proportions in the other two groups ($P < 0.01$). One-half of the top-level executives who smoked cigarettes regularly at some time during their lives have either discontinued smoking or switched to some other form of tobacco. In comparison, former cigarette smokers comprise 30.0% of the lower-level executive group and 32.7% of the nonexecutive personnel.

Discussion

There is no indication from our data that employees below the executive level exceed executives in quantity of cigarette smoking or in the prevalence of hypertension, overweight, and hypercholesteremia. The three latter conditions appear to occur at about the same rate at all levels of job responsibility. There were only three notable differences among the groups studied: (1) The mean serum cholesterol of the nonexecutives was slightly lower than that of the executives; (2) heavy cigarette smoking was proportionately more common in the lower-level executive group than in the two other groups studied, and (3) proportionately more top-level executives have discontinued regular cigarette smoking than have employees in the other job levels.

None of these differences tend to explain the lower incidence of coronary heart disease among executives. Although current cigarette consumption of the top-level executives is somewhat less than that of the other groups (Table IV), the differences are not significant and are not of sufficient magnitude to account for the twofold excess of the incidence of myocardial infarction we have observed in lower level nonexecutive employees when compared to executives. Our study, therefore, indicates that to explain this difference in incidence we must search for characteristics other than those we have examined.

Summary

Recent investigations have revealed that coronary heart disease occurs less frequently in executive personnel than among other employed persons. The purpose of this study is to find some explanation for this difference by comparing executive and nonexecutive personnel in several characteristics associated with coronary atherosclerosis: blood pressure, body

Table IV—Current Smoking Habits of Executives and Nonexecutives

Job Category	Total Respondents		Nonsmokers and Occasional Smokers		Cigarettes		Cigars		Pipes		Daily Consumption of Cigarettes: One Pack or More		Daily Consumption of Cigarettes: Two Packs or More	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Executives														
Top level	82	100.0	39	47.6	32	39.0	7	8.5	12	14.6	18	22.0	3	3.7
Other	384	100.0	159	41.4	190	49.5	20	5.2	47	12.2	114	29.7	26	6.8
All executives	466	100.0	198	42.5	222	47.6	27	5.8	59	12.7	132	28.3	29	6.2
Nonexecutives	1,033	100.0	419	40.6	492	47.6	80	7.7	104	10.1	262	25.4	33	3.2

Table V—Lifetime Smoking History of Executives and Nonexecutives

Job Category	Total Respondents		Never Smoked		Smoked Occasionally		Cigarettes		Cigars		Pipes		Two Packs of Cigarettes or More per Day	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Executives:														
Top level	82	100.0	9	11.0	3	3.7	64	78.0	11	13.4	36	43.9	5	6.1
Lower level	384	100.0	54	14.1	26	6.8	271	70.3	45	11.7	139	36.2	40	10.4
All executives	466	100.0	63	13.5	29	6.2	335	71.9	56	12.0	175	37.6	45	9.7
Nonexecutives	1,033	100.0	151	14.6	43	4.2	731	70.8	148	14.3	362	35.0	73	7.1

Table VI—Changes in Smoking Habits of Executives and Nonexecutives

Job Category	Lifetime History of Regular Smoking: No. with History of Regular Smoking	Lifetime History of Regular Smoking: Ex-Smokers No.	Lifetime History of Regular Smoking: Ex-Smokers %	Lifetime History of Regular Cigarette Smoking: No. with History of Regular Cigarette Smoking	Lifetime History of Regular Cigarette Smoking: Ex-cigarette Smokers No.	Lifetime History of Regular Cigarette Smoking: Ex-cigarette Smokers %
Executives:						
Top level	70	27	38.6	64	32	50.0
Lower level	304	79	26.0	271	81	30.0
All executives	374	106	28.3	335	113	33.7
Nonexecutives	839	225	26.8	731	239	32.7

weight, serum cholesterol, and smoking habits. The subjects of our study consisted of 1,585 male salaried employees, 40 to 64 years of age. They were divided into three categories: top-level executives, lower-level excutives, and non-executives.

No significant differences were found among the three groups with respect to the prevalence of hypertension, overweight, and hypercholesteremia, although the mean serum cholesterol of the nonexecutives was slightly lower than that of the executives. Smoking patterns were found to be essentially the same in the three groups, except that heavy cigarette smoking was more common among the lower-level executives and proportionately more top-level executives have discontinued regular smoking of cigarettes. It is concluded that none of the characteristics investigated in this study can account for the lower incidence of coronary heart disease found in executive personnel.

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Fluorides: A Critical Review

IV. Response of Livestock and Poultry to Absorption of Inorganic Fluorides

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INORGANIC compounds of fluorine are present universally in the natural environment of domestic animals. On occasion, certain phenomena in nature and certain technological activities of man have contributed unusual quantities of fluoride to the environment in specific geographic locations and situations. The hygienic problems that have appeared on such occasions have generated widespread concern as to the hazards of fluorides.[206]

The Biological Role of Fluoride in Farm Animals

Under normal conditions a small quantity of fluoride is present in the diet of domestic animals. Present evidence does not establish its essentiality in their living processes.[137]

The likelihood that small quantities of fluoride may play a beneficial role in the prevention of dental caries in livestock, as it does in man, has been explored very little. One report notes that the teeth of cattle exhibiting fluorotic lesions had a significantly low incidence of caries.[67]

Epizoology of Poisoning with Fluoride

Prior to the middle of the 19th century, fluoride was identified along with many other elements in the tissues of the body.[15-16, 19, 28, 49, 90-91]

Although biological reactions may have been observed earlier, present knowledge concerning the responses of various species of livestock and poultry to the absorption of abnormally large quantities of inorganic compounds of fluorine began with historical accounts in Iceland about the year 1000. Because of the complexity of the environment, it reasonably may be expected that the early clinical accounts include signs of diseases caused by agents other than fluoride.

Many efforts have been made during the last half century to investigate and define the signs and lesions specifically induced in livestock and poultry by the absorption of fluoride. Fluoride, in its more common inorganic forms, was fed in measured quantities and concentrations for various periods of time to cattle, sheep, and less frequently, other species of livestock.* The resultant effects exerted upon these animals, and upon animal products, were observed and sometimes measured. Unexplainable variations occurred occasionally in the susceptibility and in the response of domestic animals to apparently comparable experimental or environmental conditions. Such variations led to investigations in which the form of the fluoride, the practices in animal husbandry, the physiological status of the animals, the species of animal, and other influencing factors were controlled or taken into consideration.

History of Fluoride and Disease in Domestic Animals

Roholm[167] found early Icelandic accounts of illness (*gaddur*, spike, or *gaddjaxl* [*jaxl*, jawtooth|) and death among livestock following volcanic eruptions (although the eruptions in 1875 and 1918 were not followed by losses among domestic animals, as had occurred earlier). He reviewed the descriptions of the disease, studied the associated osseous lesions, and found elevated concentrations of fluoride in museum specimens of bones of the extremities and mandible of adult sheep that had grazed in the vicinity of the Icelandic volcano, Hekla, during its eruption in 1845. In the aggregate, the clinical descriptions, bony lesions, and concentrations of fluoride in the bones constituted, in his judgment, "proof" of a disease induced by fluoride. Although he stated that "no fluorine has ever been found in the ejecta of the

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*Cattle: references 29, 40, 47, 53, 127, 160, 165, 170, 190, 194, 204. Sheep: references 86, 88, 148, 175, 195. Swine: references 17, 18, 60, 105, 106, 107, 120, 154, 204. Poultry: references 27, 71, 72, 78, 79, 106, 108, 121, 159.

Icelandic volcanoes," he remarked that "no doubt there are gaseous fluorine compounds (HF, SiF_4) in the volcanic exhalations . . . [that] condense round the fine particles of ash together with the moisture of the atmosphere." Roholm also felt that the osseous lesions which appeared after two or more months, when he fed daily doses of sodium fluoride to sheep, were similar to those found in museum specimens of bones of the legs of sheep exposed to residues from the volcanic eruptions of 1845.

The tissues of most forage plants absorb only small quantities of fluoride even when grown where the soils and waters contain relatively high concentrations of fluoride.[166, 181, 182] However, the quantities of fluoride ion on the surface of forage and other vegetation may be greatly enhanced in areas of the world in which the soils and the waters are naturally rich in compounds of this element.[83]

Fluoride intoxication in livestock maintained under natural environmental conditions comparable to those indicated above has been reported from practically every country in the world. The ingestion of soil by animals, especially during drought, also provides significant quantities of fluoride in such environments.[83]

A disease syndrome called darmous or dermes had been recognized for many years in sheep and other livestock in North Africa (parts of Algeria, Tunisia, and Morocco), but it remained for Velu in 1922 to describe the signs and, in 1931, to establish the relationship between darmous and chronic intoxication induced by the ingestion of abnormally large quantities of fluoride in water that had passed through natural deposits of fluoride-bearing phosphorite.[168]

About the turn of the century, phosphorites (rock phosphates, acid or superphosphates, and phosphatic limestones) were being added to the diets of livestock to provide supplementary quantities of calcium and phosphorus. As early as 1902, Emmerling[57] in Germany warned against the use of lime phosphate for such a purpose in the feeding of swine, because of the observed loss of appetite, thirst, lameness, and dark and hard feces. He considered the fluoride in the phosphate to be a factor in the illness of these animals. Dammann and Manegold[48] in 1904 reported clinical signs in pigs and produced experimental evidence to incriminate the high concentrations of fluoride in calcium phosphate used as a supplement in the diet of several species of animals. In their publications in 1904 and 1914, Hart and his associates[168] in Wisconsin considered phosphorites in the diet to be beneficial to the development of the skeleton, but in 1927 they advised against the use of rock phosphate.[82] Certain early investigators recognized and reported that the addition of phosphorites to the diet led to the development of illness in livestock and poultry but did not consider the role of fluoride as the causative agent.[27, 61]

Fluoride, as hydrofluoric acid or ammonium fluoride, has been added to slops, mashes, or lees in order to control deleterious fermentation in the production of spirits. Tappeiner in 1890, (cited by Baekeland[9]) published experiments with cattle showing that "not the smallest difference has been observed in the use of such slops, either in the composition of the milk or the general condition of the animals fed upon it." However, it was remarked that the same slop should not be used more than twice in the fermentation process, if it is to be used afterward for feeding purposes, because of the accumulation of fluoride. Kötz,[110] in his review of the chemistry and physiology of fluorine in 1913, mentioned that fluoride-containing refuse (slops, lees, and mashes) from the manufacture of spirits was not used much for feed during that period in Germany because farmers found it injurious to the animals.

Early in this century reports appeared of disease in livestock in association with the pollution of air or water by fluoride-bearing wastes from certain industrial processes, with resultant increase in the quantities of fluoride in and on the vegetation, in the water, and in and on the soil. Bartolucci[11] in 1912 reported a disease in cattle grazing upon pastures contaminated by the atmospheric waste from a superphosphate-processing fertilizer plant in Italy. He associated the disease with the fluoride contained in the waste.

During the years 1911 to 1918, a disease was noted among cattle grazing in the vicinity of an aluminum works in Switzerland. Cristiani and his colleagues[45] in 1922 and later demonstrated experimentally that signs of illness, as well as certain lesions, as observed in cattle under field conditions, could be reproduced in guinea pigs by feeding forage so contaminated by various environmental and experimental means as to incriminate fluoride.[41, 46]

Hupka and Luy[101] reviewed earlier accounts and reported illness among cattle pasturing downwind from a chemical factory in Germany that used fluoride in its processes. Vegetation in the same direction also showed damage, which on the basis of chemical analyses was attributed to contamination with fluoride. Hupka and Götze also reported illness in cattle

grazing in the vicinity of a factory that processed phosphate fertilizer. The oral administration of NaF and Na_2SiF_6 to five cattle, for two to three months, caused acute and chronic illness, but not the osteodystrophic changes that had been noted in the field. These two investigators supposed that, under field conditions, fluoride injured cattle indirectly in some manner via the vegetation, and that other unidentified factors contributed to its action.[100]

The use of compounds of fluorine for the control of pests on the farm provided another potential source of exposure to fluoride. The few published cases of accidental and acute poisoning suggest that the use and misuse of these, and also of fluoride-rich fertilizer[170] and wood preservatives,[115] occasionally present a problem.

Routes of Absorption of Fluoride

The fluoride in the body of livestock and poultry is derived mainly from the ingestion of contaminated food or water. A considerably smaller quantity enters by way of the respiratory tract, being inhaled in the air, which always contains some fluoride in gaseous or particulate form. Under unusual circumstances, domestic animals may be exposed to air containing high concentrations of fluoride. As a general rule, however, the quantities of fluoride consumed with food are far greater than those taken in with the air. A single report noting the concentration of fluoride in the lungs of cattle that had been exposed to contaminated atmosphere called attention to a fivefold increase in the fluoride above the normal range.[2] However, these analytical data do not provide evidence of the exposure of the animals to fluoride in the atmosphere, since the fluoride content of the lung may be elevated whenever the other tissues of the body are relatively high in fluoride.

The systemic absorption of inorganic fluoride through the skin, under any circumstances, is negligible, although hydrofluoric acid in contact with the skin may spread directly into the adjacent cutaneous and subcutaneous tissues.

Acute Poisoning by Fluoride

Descriptions of acute illness in farm animals, in association with the ingestion of inorganic compounds of fluorine in toxic quantities, have been somewhat sketchy. This review includes cases of acute poisoning that resulted from accidental as well as experimental ingestion of fluoride (Tables I-III). It is impossible to determine the part played by the concentration of the fluoride in a specific dose, as distinguished from the cumulative effect of multiple doses, in the acute response to the absorption of fluoride, but both factors are of some importance.[14]

Acute intoxication, as produced by sodium fluoride, has been described, following the experimental administration of the salt by mouth. The most commonly observed signs were a reduction or loss of appetite,[25, 36, 37, 39, 77, 97 167] softened feces or watery diarrhea,[77, 100, 167] loss in body weight,[36, 37, 39, 163, 167] and depression or weakness.[77] Only swine vomited[77] (vomiting being extremely rare in goats, sheep, cattle, and horses). Retching was exhibited by animals that vomit rarely or not at all. Evidence of acute abdominal distress has been observed in cattle,[25, 100] salivation[77] and reduced milk yield in goats,[39] glycosuria in sheep,[73] and trembling in swine.[77]

Farm animals, after swallowing a single toxic dose of sodium silicofluoride, refused food or ate very little.[7, 50, 76, 77, 147] They developed diarrhea[50, 76, 92, 100, 112] or varying degrees of gastrointestinal distention with gas.[92, 112] Trembling was noted also.[50, 76] Swine vomited.[77]

Cattle that accidentally had ingested large but unstated quantities of almost pure Na_2SiF_6 in the diet[92, 112] became uneasy, had dilated pupils, and exhibited chewing motions with salivary frothing. Locomotion became unsteady with increasing weakness, and loss of balance when attempting to lie down. Respirations were short and rapid. The heart beat and pulse became very rapid and irregularly weak. Defecation was suppressed and urination scanty and infrequent. Convulsions appeared with increasing frequency followed by death in 12 to 14 hours. A few cattle developed milder signs on the second day, seemingly relieved by subcutaneous injection of caffeine but required destruction because of serious "cardiac disturbances" at 12 to 14 days.

Several cases of poisoning have been reported among sheep and cattle on farms where fluoride-containing superphosphate, used as fertilizer, had been stored within reach of the animals.[170] Acute progressive signs in one cow and one sheep were reported as follows: The animals drank water ravenously, ground their teeth together, and salivated. They refused food. Palpation in the region of the abomasum evoked evidence of pain. The mucous membranes of the eyes and mouth became hyperemic. Defecation occurred frequently, the feces contained streaks of mucus, and developed into a profuse bloody diarrhea. Body movements appeared to be difficult. The animals lay with extremities extended and spread widely. Tonic convulsions developed, with general weakness

and accelerated respirations. Terminally the body temperature dropped below normal, while the pulse became thready and rapid.

Pigs given 304 to 380 mg of fluoride (as a contaminant of calcium phosphate [8.4% of NaF]) daily, in their food, refused further food on the sixth day, exhibited muscular weakness and, subsequently, paralysis. One pig died on the tenth day, and the other, although its food was no longer contaminated, lost in weight persistently. One pig, given food containing 190 to 304 mg of fluoride daily, for 22 days, vomited on the tenth day, took no food for several days and continued to lose weight for several weeks after the food was no longer contaminated. The visible mucous membranes were inflamed, and the feces were hard, dark brown, and streaked with gray-green mucus.[48, 57]

A horse refused to eat on the third day after having ingested 3.4 to 3.8 gm of fluoride (in calcium phosphate) in its feed on each of the previous two days. The body temperature rose about one degree. The pulse weakened and almost doubled in rate, while the respiratory rate was normal. The animal had an hour-long episode of colic and restlessness, following which there was recurring severe diarrhea (containing an occasional hard mass of feces). The urine was dark yellow in color and alkaline in reaction, with traces of albumin. The blood appeared to coagulate poorly and to exhibit some hemolysis.[48]

A somewhat apocryphal account is given of an accident in which a horse fell through an iron cover and into a drainage ditch of a fertilizer plant, where it inhaled H_2SiF_6 vapors for about 10 minutes before it could be removed. Shortly thereafter it showed signs of unrest, perspired freely, and had difficulty in breathing. An hour later the hindquarters became paralyzed, and after four hours it died.[3]

Acute Dental Changes

Investigations using radioisotopes have provided the most direct evidence of the early changes induced

Table I—Response of Domestic Animals to Various Levels of Fluoride Dosage: Minimal Response

COMPOUND	SPECIES OF ANIMAL	CONCENTRATION (F^- ppm)	DAILY DOSE (mg F^-/kg body weight) or mg F^- per animal	SCHEDULE	REFERENCE
ACUTE SIGNS NOT INDUCED:					
NaF	Cattle	300 and less	~4.12 mg/kg and less	in diet, several months	97
	Swine	~7.0 to 90.5	———	in feed, ad lib 75 days	36, 37
	Sheep	———	1.83 and 2.35 mg/kg	in capsule orally, 1 time	153
	Horse	———	13.8 to 41.4 mg/kg	" "	77
		———	11 mg/kg	in capsule, every other day, 3 doses	
Na_2SiF_6	Cattle	~196 to 982	196 to 982 mg per animal	in molasses and bran, 1 time	50
	Sheep	24,249	12 g per animal	in bran (refused feed), force fed, 1 time	147
Ca Phosphate (2-3 percent NaF)	Horse	———	1.14 to 1.9 g per animal or 2.54 to 4.22 mg/kg	in the diet, for 11 days	48
$3NaF \cdot AlF_3$ (synthetic cryolite)	Chicken	———	5000 mg/kg	orally, 1 time	62
	Cattle	10,860 and 27,150	25.3, 38.8, and 51.2 mg/kg	daily in water, orally 28, 30, and 15 days resp.	168
	Sheep	———	599 mg/kg	in capsule, 1 time (1 sheep)	77
AlF_3	Swine	20, 361	469 and 1730 mg/kg	in feed 1 day (2 pigs)	77
$Al_2(SiF_6)_3$		14, 246	1210 mg/kg	" (1 pig)	
BaF_2		4, 334	178 mg/kg	" "	
$3NaF \cdot AlF_3$		27, 145	919 and 1196 mg/kg	" (3 pigs)	
MARGINALLY ACUTE SIGNS INDUCED:					
NaF	Cattle	302 to<453	0.9 to<136 g per animal	daily in water, stomach tube, several days	25, 100
		2,262 and 6,786	5.27 and 12.8 mg/kg	in water, orally, once daily 28, and 15 days, respectively	168
	Horse	———	10.3 mg/kg	in feed, daily, during 12 days omitting day 6 and 11	77
		———	19.9 mg/kg	in capsule, daily for 2 days	
		———	7.9 mg/kg	in feed, daily for 12 days	
Na_2SiF_6	Cattle	403 to<909	1.2 to<1.82 g per animal	in water by stomach tube for 2 to 3 months	100

Table II—Response of Domestic Animals to Various Levels of Fluoride Dosage: Acute Response

COMPOUND	SPECIES OF ANIMAL	CONCENTRATION (F^- ppm)	DAILY DOSE (mg F^-/kg body weight) or mg F^- per animal	SCHEDULE	REFERENCE
ACUTE SIGNS INDUCED:					
NaF	Cattle	600 and more	~6.13 mg/kg or more	in feed for 2 to 3 days	97
		453 to 903	1.36 to 2.71 g per animal	daily in water, stomach tube, a few days	25, 100
		———	37.5 mg/kg	in grain feed, 1 time	77
		13,572	23.6 mg/kg	in water, orally, once daily for 27 days	168
	Swine	4,524 and more	———	in feed for 1 to 3 days	77, 134
		18,096	153 mg/kg	in feed, for 1 meal	77
		>90.5	———	reduced intake of feed, 75 days	36, 37
	Sheep	———	136 mg per animal	in mixture of milk and water, orally, 1 time	73
		———	" "	" "	73
	Goat	4,524	11.9 and 12.6 mg/kg	in water, stomach tube, daily 8 days	39
		———	73.7 mg/kg	in capsule, 1 time	77
		———	166 mg/kg	in grain feed, 1 time (refused feed)	
	Horse	———	46.0 and 55.2 mg/kg	in feed for 1 day	77
		———	22.6 g per animal	in grain feed, 1 time (refused feed)	77, 115
	Chicken	6, 786	———	in feed for 10 days (most of feed refused)	77
Na_2SiF_6	Cattle	1,820 and 29,200	0.54 and 8.59 g per animal	in water, orally, 1 time	50
		909 to 1,212	1.82 to 364 g per animal	in feed for a few days	100
		———	~1.22 to 2.43 g per animal	in distilled water for a few days	93
	Swine	———	121 to 668 mg/kg	in feed for 1 meal	77
	Goat	606	———	in feed for 4 days	7
		———	69 mg/kg	in feed for a few days	
Ca Phosphate (2 - 3 percent NaF)	Swine	———	190 to 304 mg per animal	in feed for 22 days	48
		225.8 and 248.8	———	in feed for a few days	
	Horse	———	3.4 g per animal increased to 3.8 g	lower dose (=7.57 mg/kg) increased after 2 days to higher dose (=8.44 mg/kg) for 2 days	48
$CaSiF_6$	Cattle	1,250 and 2,500	1.25 and 2.5 g per animal (intended)	in feed (most of feed refused)	190
$3NaF \cdot AlF_3$ (synthetic cryolite)	Cattle	43,400 and 54,300	464, 639, and 944 mg/kg	in water, orally, once daily, 16, 76, and 27 days, respectively	168
BaF_2	Swine	6,501	267 mg/kg	in feed for 1 day (1 pig)	77

Table III—Response of Domestic Animals to Various Levels of Fluoride Dosage: Lethal Doses

COMPOUND	SPECIES OF ANIMAL	CONCENTRATION (F^- ppm)	DAILY DOSE (mg F^-/kg body weight) or mg F^- per animal	SCHEDULE	REFERENCE
LETHAL DOSE					
NaF	Cattle (calf)	———	45.2 mg/kg	orally, 1 time	64
	Swine	22,620	293 and 554 mg/kg	in feed for 1 day	77
	Sheep	———	86.6 and 129.8 mg/kg	in capsule, 1 time	77
		———	15 mg/kg	in feed for 51 days	167
	Goat	———	99.5 to 199 mg/kg	in capsule, 1 time	77
		———	285 mg/kg	in feed for 1 day	
	Horse	———	>45 g/kg	orally, 1 time	115
Na_2SiF_6	Sheep	24,248	24.1 g per animal	in bran (refused feed, so force fed), 1 time	147
		———	334 mg/kg	in capsule, 1 time	77
	Goat	———	1.82 g per animal	in feed for 1 meal	7
		———	82 to 90 mg/kg	in feed for a few days (calculated)	
Ca Phosphate (2 - 3 percent NaF)	Swine	———	304 mg increased to 380 mg per animal	in feed (low dose = 24.4 mg/kg for 4 days increased to 30.4 mg/kg for 5 days)	48
$BaSiF_6$	Chicken	———	27.1 mg/kg	on grain, 1 time	104
$3NaF \cdot AlF_6$ (synthetic cryolite)	Sheep	———	1 g/kg	orally, 1 time	62

in the teeth of animals by fluoride. This technique has furnished the earliest approach to the recognition of dental and skeletal lesions, and has provided means for investigating the types and the mechanisms of such lesions.

Belanger and co-workers[14] investigated the effect of the taking of a diet contaminated with toxic quantities of sodium fluoride for 40 to 75 days upon the growth of the incisors of pigs, by means of autoradiographic visualization with Ca^{45}. The dentin formed during the period of high fluoride intake appeared less dense (radiopacity) than normal. Other abnormalities were (*a*) a granular appearance of the dentin, interpreted as representing a variation in the mineral pattern (abnormal crystalline deposition, or at least abnormal crystalline arrangement), as the basis for the irregular outline of the dentinal tubules; and (*b*) serration at the edge of the proximal portion of the dentin that seemed to indicate a deficiency in the dentin-forming mechanism. This abnormality was more apparent in areas of more intensive growth. The retarded migration of the Ca^{45} band and its wavy appearance appear to have resulted from a disturbance both of the formation of dentin and of the mineral pattern of the teeth during the ingestion of toxic quantities of fluoride.

The regular appearance of an autoradiographic band after three days of taking a diet containing 1% of NaF indicated that time was a factor in the production of these changes. There was no evidence of hypermineralization in the presence of the disturbed mineral pattern. The incisor teeth of pigs fed (ad libitum) on diets containing fluoride (as NaF) in concentrations of 90.5 ppm did not exhibit any significant abnormalities. However, a concentration of fluoride greater than 90.5 ppm (the same total intake of fluoride in a smaller quantity of feed) was associated with a retarded growth of dentin and cementum and an irregular wavy pattern in the radioactive band over the dentin. At the level of 452.4 ppm in the diet of the pigs, there were marked retardation in the growth of dentin, cementum, and alveolar bone and a coarse wavy pattern of the autoradiographic bands.

Pigs, fed from the time of their weaning to approximately six months of age on diets containing fluoride in concentrations between 2.24 and 22.4 ppm, exhibited defects in the formation of enamel, ranging from a complete break in the contiguity of the enamel to thinning or gnarling. The region which appeared to be most susceptible was about a third of the distance from the occlusal surface on the buccal aspect of the second molar teeth. Some defects were noted on the occlusal surface. The lesions appeared only once in five animals on the first molar tooth.[58]

Pathology in Acute Intoxication

The gross findings at necropsy in cases of acute poisoning in a small number of cattle, sheep, goats, swine, horses, and chickens included focal or general congestion and submucosal hemorrhages in the gastrointestinal tract. This was observed in acute poisoning due to NaF,[77] Na_2SiF_6,[77, 112] superphosphate,[170] and Ca phosphate[48] (the latter two contained fluoride as a contaminant). In addition, hemorrhages were found throughout the internal organs of goats. One pig showed marked congestion of the liver and kidneys.[77]

Cattle accidentally poisoned with sodium fluosilicate showed some mucosal inflammation and blood-tinged fluid in the small intestines and an increased quantity of pericardial and intracranial fluid,[92, 112] together with pulmonary emphysema.[170] Hyaline material was found in the hepatic and renal cells, together with hyaline casts in the renal tubules of swine poisoned with sodium fluoride.[1]

Comar and co-workers[37] described a reduction in the growth of endochondral bone in pigs, fed for 40 and 75 days, respectively, on diets containing fluoride (as NaF) in concentrations greater than 90.5 ppm, and 452.4 ppm. A distortion of the linear pattern of hypertrophied chondrocytes was visualized by autoradiography as early as the tenth day after the intravenous administration of Ca^{45}, following a period of 30 days of feeding a contaminated diet. The distorted pattern did not appear when the growth of endochondral bone was limited by a reduction in the intake of feed alone (a reduced intake of feed had resulted from the contamination of the diet with fluoride).

This suggested to these workers that the collagen fibrils did not provide adequate support for the cartilaginous columns, because of changes either in themselves or in the medium in which they were embedded. The labeled calcium (Ca^{45}) originally deposited near the epiphyseal plate disappeared so rapidly from the primary and secondary spongiosa as to point to an acceleration of the normal resorptive process. There was some suggestive evidence of a decrease in the size of the cartilaginous cores in the trabecular bone (secondary spongiosa), and definite evidence of an increased osteoid border. The matrix was devoid of Ca^{45}, in evidence either that the osteoid tissue produced was not able to hold Ca^{45} or that the process of Ca^{45} deposition was disturbed.

Metabolic Phenomena

The only radioactive isotope of fluorine, F^{18}, has a half-life of 112 minutes, making it requisite that all work involving its use be completed within 10 hours. This characteristic has limited sharply the use of this technique in the elucidation of the metabolism of fluoride.

Ca^{45} and also Na^{22} have rarely been used in conjunction with tagged or untagged fluoride in attempts to visualize the metabolic pathways of calcium and sodium in the presence of elevated quantities of fluoride. Additional relationships in the metabolism of fluorides could profitably be explored with radioactive tracers such as P^{32}.

An investigation by Perkinson and co-workers,[153] using F^{18}, provides direct evidence of the movements and disposition of fluoride ions very shortly after their administration in low concentrations to sheep and cattle. Sodium fluoride (F^{18}) was found in the bloodstream of a sheep as early as five minutes after its oral administration in a capsule. The concentration of fluoride (F^{18}) in the blood reached the maximum at 180 minutes and decreased thereafter. In a cow, the peak concentration of F^{18} occurred in about 5 hours, as compared with that of Ca^{45}, which has been reported to require about 40 hours. It may be that the fluoride was absorbed in the upper digestive tract, while the calcium was absorbed principally in the lower tract.

Two hours after the oral administration of F^{18} to a cow, practically all of the dose was in the rumenal contents, and less than 1% was found in the remainder of the gastrointestinal tract. Two hours after intravenous injection, about 5% of the F^{18} was found in the gastrointestinal tract, of which 4% was in the rumen and reticulum, 1% in the large intestine, and minor amounts in the other parts of the tract.

Autoradiograms of bones of several sheep, given F^{18} in a capsule, exhibited a typical pattern of deposition; there was heavy deposition in the primary spongiosa below the epiphyseal plate, generalized spotty distribution in the trabecular region, spotty but linear deposition in the endochondral region, and relatively light deposition along the endosteal and periosteal surfaces. The pattern suggested that the deposition of F^{18} can be accounted for by ion exchange, probably with the hydroxyl or bicarbonate ions of the apatite structure, rather than by incorporation into newly forming crystals of bone. This is consistent with the idea that such physiological factors as vascularity, blood supply, and crystal size will govern the relative deposition of F^{18} by the exchange process.[153] Interpretations of these findings must take into account the exchange reactions occurring.

An autoradiogram of the mandible exhibited a relatively heavy deposition of F^{18} in and around the enamel of an unerupted molar, a less darkened area in the pulp cavity, "probably due either to the soft tissue level or overlapping of skeletal tissue in the section."

Determinations of the cell to plasma ratio of F^{18} in 23 samples of whole blood from sheep yielded the average of 0.66 ± 0.04. In one minute after the intravenous injection, the ratio was fully established, in evidence of the rapid penetration of fluoride into the erythrocytes.[153] This partition resembles closely the relationship between the water content of the cells and that of the plasma in the blood of sheep.

The recovery of F^{18} per gram of fresh material from various parts of developing eggs varies from nil to 0.02% of the dose injected into the bloodstream of laying hens. In general, the concentration of F^{18} was greater in the

less mature yolks. The highest value occurred in an incompletely calcified shell, and other high values were found in fully calcified shells. With one exception, the concentration of F^{18} in the albumin was higher than in the yolk.[153]

The portion of F^{18} recovered in milk, collected over the period of 680 minutes after the administration of a single oral dose to a cow, amounted to 0.018%. The peak concentration in milk was reached in between 200 and 300 minutes, while in the blood it was reached in about 100 minutes. Following an intravenous injection, 0.19% of the dose was recovered from milk collected during a period of 609 minutes.[153]

Pregnant cattle, given a series of daily oral doses of sodium bifluoride and then of sodium silicofluoride, with acute toxic manifestations during each series followed by recovery, did not exhibit changes in the concentrations of Ca and P in their blood serum.[93] Lambs and goat kids developed intense glycosuria within two hours after the ingestion of a single large dose of sodium fluoride.[73]

Fluoride in Tissues Following Brief Periods of Absorption

Ribs taken by biopsy from Holstein heifers, after they had been fed for 28 days on diets to which fluoride (as NaF) had been added in various concentrations, contained fluoride in the concentrations tabulated below.[185]

F (as NaF) in Diet, (ppm)	Concentration of F (ppm) in fat-free, dried rib
14	avg. 355 (240-500)
28	avg. 386 (290-470)
50	avg. 666 (560-840)
94	avg. 1244 (1075-1400)

At the end of 28 days, sheep given fluoride (as NaF) in their diet, in concentrations ranging from 6 to 206 ppm, had fluoride in their tissues in the concentrations given below.[97]

Concentrations of Fluoride in Tissues in ppm		Average Findings in Tissues of Control Sheep
Metacarpal	285-1,650	....
Mandible	345-3,400	....
Liver	0.16-0.63	0.89
Heart	0.14-0.92	1.5
Kidney	0.43-1.42	3.1
Lung	0.05-0.54	0.92
Spleen	0.05-0.71	0.92

Neither of these series of results can be regarded as indicative of those which would be found in animals to which a single large dose of fluoride has been administered.

Excretion of Fluoride in Acute Intoxication

An elevated concentration of fluoride was found in the urine of a horse 20 minutes after the oral administration of a toxic dose of sodium fluoride.[115] The specific gravity, pH, and fat content of the milk of acutely poisoned goats was altered (associated with depressed lactation and refusal of feed).[39]

Influence of Sublethal Intoxication on Pregnancy

There is no evidence to indicate that acute sublethal doses of fluoride affect directly the ability of pregnant animals to reach term and to deliver normal young.[25, 93] There is some indication that the effect upon food intake may be serious enough to inhibit the production of milk during the suckling period, thus affecting the growth and development of vigorous young, and, if continued long enough, leading to cachexia in the female and indirectly to an irregular estrus or anestrus.[60]

Therapy of Acute Poisoning by Fluoride

There is no evidence in the literature of the development of a satisfactory or certainly beneficial treatment for animals that have been poisoned by fluoride, aside from symptomatic or supportive measures.

The treatment of three cases of acute poisoning in cattle that had ingested superphosphate fertilizer (containing fluoride), has been described.[170] Subcutaneous injections of caffeine were administered, and followed by the oral administration of hot milk and egg white in water. A strained gruel of linseed and barley was given for five days. The animals recovered. That the therapy was responsible for this outcome is not self-evident.

Levenshtern and Dubrovin[115] considered intravenous injection of calcium chloride with 25% glucose solution, as the most effective antidotal treatment of acute poisoning in conjunction with: gastric lavage using tannic acid solution, 2% soda bicarbonate solution; internally, a laxative or soda with milk; cardiac signs, caffeine and camphor.

In view of the lack of evidence in support of the opinion that the mechanism of intoxication by fluoride, even in its most acute form, is that of the removal of calcium from its customary biochemical relationship in the animal organism, the foregoing recommendation does not command enthusiastic attention. There are no reports indicative of the efficacy of these therapeutic procedures.

Note: The conclusion of Section IV of this Topical Review, including all references for the Section, will appear in the November, 1961, issue of this journal.

(The author's address is Kettering Laboratory, University of Cincinnati College of Medicine, Eden Ave., Cincinnati 19, Ohio.)

Case Report

Blindness in a Fur Worker

Donald J. Lyle, M.D., and Mitchell R. Zavon, M.D., Cincinnati, Ohio

THERE ARE reports in the literature on the onset of blindness in association with a history of exposure to carbon tetrachloride. The following case is presented as an example of this association. Unlike most of the cases in the literature,[1-6] reasonably accurate exposure data are available in this instance.

Case History

A 48-year-old white man with no known physical disabilities was seen in 1953. At that time his vision was better than 20/20 in each eye with corrective lenses (O.D. +0.75 sph + 0.75 cyl × 3; O.S. same correction × 170).

In February, 1958, the patient complained of failing vision in his left eye. Examination revealed paresis of both sixth cranial nerves and marked horizontal nystagmus of the abducted eye. There was poor reaction of both pupils to light but perfect accommodation. A questionable haziness was noted on the nasal border of the right disc, with the possibility of slight paleness of the left disc, though the disc borders were sharp. There was a punched-out defect in the left fovea with a denuded choroid about ⅛ disc diameter, surrounded by retinal edema. There was no retinal detachment. Retinal arterial pressure was slightly higher on the left disc than on the right disc. Vision was recorded as O.D. 20, 30+; O.S. finger counting, central scotoma.

Findings on x-ray films of the skull were within normal limits, as were results of serologic and spinal fluid examinations.

In September, 1958, the right eye began to fail. Vision in this eye was limited to counting fingers at 30 cm. A punched-out area in the right fovea, similar to that previously described in the left fovea, was now observed. Paresis of both external recti (VI) was still present as was the nystagmus. Central scotomata were now found in both visual fields. Peripheral fields were normal.

In September, 1959, no symptoms were present except loss of vision in both eyes. There was no evidence of paresis or paralysis of extraocular or intraocular muscles. There was no pain on eye motion. Pupils were equal, and all reflex reactions were normal. Corneal reflexes were normal. Examination of the external segments of both eyes with the anterior segment microscope and slit lamp revealed nothing abnormal. The fundus showed slight pallor of the nerve heads and possibly some edema of the macular area. Vision in the right eye was limited to counting fingers at 122 cm (4 ft.) and in the left eye to counting fingers at 30 cm (1 ft.). Visual fields showed a normal peripheral extension with absolute central scotoma in each eye. Intraocular tension was normal.

Diagnosis at that time was optic neuritis with involvement of the macular axons of both eyes. Treatment was limited to use of corticotropin (ACTH). Another ophthalmologist, shortly after this date, advised that the patient stay "away from fumes, smoking, and drinking" on the assumption that the neuritis was "on a sensitivity basis," and re-

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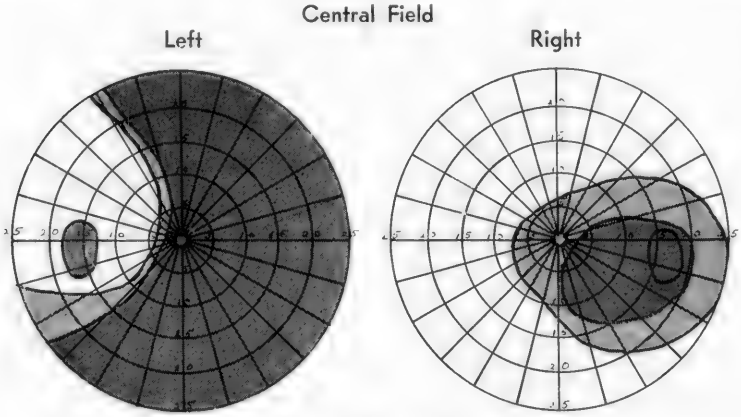

Light gray = 3 mm white test object
Deep gray = 5 mm white test object

Illumination—good; Cooperation—fair; Reliability—questionable

strict salt and fluid intake—general edicts issued by many physicians on the basis of previous experience with similar situations.

At the time of writing (February, 1960), there has been slight improvement in the central fields of the right eye. There has been no change in the left eye.

Work History

From June 1935 until August 1959 K.B. was employed as a fur cutter and supervisor of several large fur storage vaults. He did no fur cleaning but examined all the finished work. During this period the furs were fumigated with a mixture consisting of carbon tetrachloride (85.1%), ethylene dichloride (12.1%) and ethylene dibromide (2.8%).

Fumigation of the furs was performed by pumping the fumigant into partially covered pans on the floor of the fur storage vaults, which are of either 25,000 or 50,000 cu. ft. capacity. Fumigation is performed twice each year in each vault by blowing air across the fumigation pans. In the course of his work the patient would enter the vaults from time to time during fumigation but not while the most intense concentration of fumigant was present. The time in the vaults was minimal and apparently measurable in minutes at each visit. At no time did he enter the vault sooner than one hour after the fumigant had been added to the pans. The data shown in the table indicate that the level of carbon tetrachloride did not exceed 2.3 ppm nor did the level of ethylene dibromide exceed 0.35 ppm.

Environmental Findings

It was not possible to determine the concentration of fumigant in the vicinity of the fur coat after fumigation. The results of the sampling are shown in the accompanying table.

Comment

This case is another instance of the association of blindness with exposure, however minimal, to carbon tetrachloride. Careful review of the cases of similar nature presented in the literature also indicate an association, but in no instance do they prove a cause-and-effect relationship. The animal studies bearing on this problem were done many years ago and would bear repetition. On the basis of this case and similar case presentations in the literature, it is impossible to say that carbon tetrachloride causes blindness.

Environmental Findings

Time of Sampling	Carbon Tetrachloride*	Ethylene Dibromide*
	ppm	ppm
One hour after fumigant was added to the pans	1.3	<0.1
Eighteen hours after fumigant was added to the pans	2.2	<0.1
One hour after fumigant and vault had been cleared of vapors	1.1	<0.1
Twenty-nine hours after fumigation	2.3	0.35

*Threshold limit value for each, 25 ppm.

POSTSCRIPT: A refraction on June 13, 1961, after this report was submitted for publication, gave the following values: O. D. 20/100, corrected 20/25; O. S. 20/200, corrected 20/35.

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U. S. PUBLIC HEALTH SERVICE DIVISION OF OCCUPATIONAL HEALTH

will conduct a three-day course on

Training for Occupational Health Nurse Consultants

November 28-30, 1961

The course is designed to increase the knowledge, skills, and abilities of occupational health nurse consultants in government agencies. Covered in the course will be counseling techniques and nursing education research. Applications for the course or requests for information should be addressed to the Chief, Training Program, Robert A. Taft Sanitary Engineering Center, 4676 Columbia Parkway, Cincinnati 26, Ohio, or to a Regional Office Director of the Department of Health, Education and Welfare.

Industrial Medical Association

THE PRESIDENT'S MESSAGE

GRADIE R. ROWNTREE, M.D.

TODAY we should like to emphasize the importance of the function of the district counselor in furthering the goals of the Industrial Medical Association. No national-type program such as that of IMA can be carried forward effectively "from the top" alone. Unless there is strong local interest and activity at what we might call the "base of the pyramid" (the local organizational structure), such a national endeavor is likely to be ineffectual.

The district counselors are a thoughtfully selected group of dedicated IMA members appointed annually by the President of the Association to represent it in their respective districts. There are more than 60 such counselors in the United States, Canada, and Puerto Rico. Each generally represents one state or province, but, because of geographic or population considerations, occasionally more than one counselor is appointed in such an area. Reappointment after a one-year term is reserved for those who have served with unusual capacity, energy, and devotion.

If we consult the statement of responsibilities of these counselors as outlined by Dr. Edward J. Schowalter, and relate it to some specific activities by and between these physicians and local IMA membership as such, I believe it will suggest much that can be done to further our national goals.

For example, in his function of maintaining liaison with members, mutually interpreting national IMA policies and services and members' attitudes and activities, the counselor is a resource person to assist with ideas and plans and in meeting occupational medicine needs in your community.

Again, as he solicits recommendations for IMA membership applications, the counselor needs local members' suggestions. This applies similarly for recommendations in the areas of fellowships, awards, committee appointments, as well as elective office prospects in the Association.

The counselor, as you may know, provides requested guidance in referring professional questions or problems to members particularly qualified to deal with them at local or national levels. And members should feel free to take advantage of such help. Likewise, as the counselor cooperates with the component society in plans for regional meetings and year-round activities, encouraging members to participate in and support them, the members should respond. Any member having an interesting case, a research project, or a worth-while observation on any aspect of occupational medicine should make this known to the district counselor or officers of the component society for possible inclusion in a program.

Especially in areas where there is no component society, the district counselor carrying out his duties needs assistance from IMA members in keeping alive county and state medical society committees on occupational medicine. Where there is no such committee in an appropriate society, the IMA membership can help the counselor to get one established.

The counselor's duties as an official representative of the national IMA organization include submitting annual activity and progress reports and special reports where warranted, making surveys as requested for Occupational Health Institute accreditation, stimulating interest by qualified district members for service on standing committees or in specialized assignments, encouraging companies in his district to start industrial health programs and the like, to all of which the local IMA members can make essential contributions.

Thoughtful cooperation with the district counselors affords a continuing way for IMA members to promote the national program to which we all are dedicated.

Book Reviews

THE FOOT AND ANKLE—THEIR INJURIES, DISEASES, DEFORMITIES AND DISABILITIES. Philip Lewin, M.D. Ed. 4. Price, $14.00. Pp. 612, with illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, Pa., 1959.

By TED J. VICKMAN, M.D.

The author's declared purpose is to present a work on the foot and ankle that will tell the general practitioner what to do for the common conditions in and around the foot; that purpose is fulfilled within the limitations of an unfortunate lack of organization. Further, many procedures are described that are demanding of skills and experience that place them in the category of special foot surgery. Their inclusion may help the nonspecialist to understand the foot better but likewise may tempt him to undertake procedures fraught with technical pitfalls.

In this fourth edition of his book devoted entirely to the foot and ankle, Lewin has expanded and up-dated, to a limited degree, a work that is somewhat tradition-bound insofar as his basic approach to evaluation and treatment are concerned. The book is, in fact, a quaint, but exhaustive, compendium of pedal miscellany, reflecting a broad experience and a venerable wisdom.

An initial chapter on the anatomy of the foot and ankle appears to be an obligatory inclusion and does not succeed in imparting an understanding of the structural peculiarities and pathodynamics of the extremity, and provides no system of surgical approach to the structures involved.

A chapter on basic principles of foot and ankle disturbances is devoted largely to history taking, detailed examination from a foot specialist's point of view, and electrodiagnosis.

Ambulatory treatment of static and traumatic conditions is detailed in a separate chapter, covering adhesive strapping, techniques of felt pads, and prescription of shoe modifications.

The remaining chapters cover, in varying degrees of detail, congenital anomalies, static deformities, and various disorders, classified according to regional anatomy, anatomic structure, etiology, and circumstance of injury. Each subject is treated separately, with considerable overlapping of subject matter, and throughout these chapters are miscellaneous insertions that seem to defy classification and, therefore, indexing and ultimate use of the information when it is needed.

There is a sizable appendix brimming with pertinent material of wide range, from regional anesthesia to phasic activity of the leg musculature, and including numerous useful surgical techniques, consistently miscellaneous in its presentation.

Dr. Vickman is in the practice of orthopedic surgery in San Francisco, Calif., and is on the staffs of St. Francis Memorial, Children's and the University of California Hospitals, among others.

Of particular interest, reflecting his general trend of presentation and subject matter, is a sizable list of "Footnotes and Footsteps—Pedigrams —Wise Words," a collection of adages, axioms, chestnuts, and a poem called "Old Shoes."

In summary, Dr. Lewin's book contains a nearly comprehensive collection of information regarding the foot and ankle reflecting a large experience, best used after a thorough initial reading, poorly suited as a reference or text owing to an unfortunate lack of organization and practical indexing.

Publications Available

The following list of publications has been compiled on the basis of probable interest to the readers of this Journal and is presented solely for the purpose of giving notice of their availability.

Health Care for California: A Report of the Governor's Committee on Medical Aid and Health. Price, not given. Pp. 104. Published by the State of California Department of Public Health, 2151 Berkeley Way, Berkeley 4, Calif. December, 1960.

This report constitutes a thorough examination by a committee appointed by the Governor of California of the problems associated with providing health and medical care for an expanding population. The study considers problems associated with organization and distribution of health services, means of payment for health services, health manpower problems, and the spectrum of health services available.

Eating Is Fun . . . For Older People, Too. Price, single copy, $.50; quantity discounts available. Pp. 28. American Dietetic Association, 620 North Michigan Avenue, Chicago 11, Illinois.

This small pamphlet is designed for the use of managers of small nursing homes and other homes devoted to the care of the aging. Its contents are also applicable to use by individual families. Many menu patterns are given. These are based upon normal nutritional requirements of older persons. There is also information on modified diets, a section on tips on saving money as well as a section on training employees with respect to sanitation and hygiene of foods.

In-Plant Medical Programs in New Jersey. Bernard Goldstein and Jack Chernick. Price $1.00. Pp. 50, with 5 charts. Institute of Management and Labor Relations, Rutgers—The State University, New Brunswick, N. J. (To order copies, write to Library, Institute of Management and Labor Relations, Winants, Rutgers—The State University, New Brunswick, N. J.)

This is a report of a survey of in-plant occupational medical programs in New Jersey. The study began in September, 1958, and has been concerned with the scope, functions, and utilization of such

programs. It includes information on medical personnel, medical facilities, medical services, and related programs. There is also a section on the status of occupational health programs in the construction industry in New Jersey.

Questions

The questions published in this column are edited versions of requests for information that have been received during recent months. The answers have been prepared by one or more competent authorities in the particular area covered by the question. Readers may address questions to the Editor.

Question: *What is the mechanism of toxic action of organic cyanide?*

(EDITOR'S NOTE: Following the appearance of two articles in this journal on the toxicity of acetonitrile, the above question was presented to certain individuals whose opinions follow. Their comments constitute a difference of opinion with those set forth by the authors of the original articles. Dr. Marvin Amdur, author of one of the articles, was invited and accepted the opportunity to reply in rebuttal. The following are comments which should be of interest to readers in reaching an answer to the above question.)

In the December, 1959, issue of The JOURNAL OF OCCUPATIONAL MEDICINE, Marvin L. Amdur, M.D. (p. 627), and U. C. Pozzani, M.D., and colleagues (p. 634) express the opinion that the mechanism of action of acetonitrile is not necessarily directly related to the formation of inorganic cyanide. Amdur postulated that the clinical manifestations exhibited by his patients who suffered acute exposure were due to thiocyanate rather than cyanide. He based his conclusion on the delayed response and on the similarities of the clinical symptoms and signs observed in his cases and those described in the literature as characteristic of thiocyanate intoxication. Pozzani et al. expressed the belief that delayed death in dogs following exposure to acetonitrile was caused "at least in part, by the formation of cyanide in vivo." They stated, however, that concentrations that are high enough to cause rapid death are lethal because of an undefined toxic action of the intact acetonitrile molecule, possibly inhibition of liver enzymes. The latter concept is based on the assumption that inorganic cyanide could not have been formed rapidly enough to reach concentrations high enough to produce rapid death.

The following different opinions are offered: Acetonitrile has practically no local toxic effects in higher animals (Meurice, J.: *Arch. internat. pharmacodyn.* 7: 11, 1900); it has been compared to the anesthetics of the fatty acid series (Giacosa, P., cited in *Malys Jahresber. Tierchemie* 15: 93, 1885), and its general anesthetic action has been reported as weaker than that of ethanol (Overton, E.: Studien uber die Narkose, Jena, 1901, p. 105). As far as inhibition of enzymes is concerned, only relatively very high concentrations interfere with enzymatic action or tissue respiration in vitro. For instance, 4% barely inhibits indophenoloxydase and decreases respiration of avian red cells by 30 to 70% (Gerber, C.: *Compt. rend. Soc. Biol.* 71: 45, 1911; Vernon, H. M.: *Biochem. Ztschr.* 51: 1, 1913; Warburg, O.: *Ztschr. physiol. Chem.* 71: 479, 1911; Warburg, O., and Wiesel, R.: *Arch. ges. Physiol.* 144: 465, 1912). These reports indicate that the intact acetonitrile molecule has a low intrinsic toxicity.

In a study of the formation of inorganic cyanide from aliphatic nitriles in rabbits (Rieders, F.: *Fed. Proc.* 11: 386, 1952), the following results were obtained:

All the normal aliphatic mononitriles from C_2 to C_6, i.e., from acetonitrile to capronitrile, gave rise to inorganic cyanide in the blood with *subsequent* prolonged increase of thiocyanate levels in the blood. The rate at which cyanide was split off increased markedly from aceto- to butyronitrile and then decreased somewhat with the higher homologues; it was practically linear with time at any one dose but rose markedly with increasing doses. The highest concentration of inorganic cyanide attained in the blood was found to be a direct function of the rate of formation during 7- to 10-hour observation periods—i.e., the faster the rate, the higher the level of inorganic cyanide in the blood.

At clinically toxic dose levels of these nitriles, some or all of the following phenomena were observed, paralleling increasing concentrations of inorganic cyanide in the blood preceding the appearance of significantly increased thiocyanate levels in the blood: labored breathing followed by shallow respiration, restlessness, tonic-clonic convulsions, prostration, hypothermia, and rales.

Thus, since the signs depended on and paralleled inorganic cyanide concentrations but not thiocyanate concentrations and since the signs were characteristic of those seen in slow infusions of cyanide (Rieders, F.: *J. Pharmacol. & Exper. Therap.* 110: 43, 1954), the toxicity of aliphatic saturated nitriles, including that of acetonitrile, was considered fully explicable by cyanide formation.

The toxicity of thiocyanate is problably due to its biotransformation to cyanide. This becomes of acute toxicologic significance only at thiocyanate dosages that result in blood levels of thiocyanate in excess of therapeutically desired levels (8 to 12 mg/100 ml blood) (Goldstein, F., and Rieders, F.: *Am. J. Physiol.* 167: 47, 1951). This biotransformation most probably accounts for some of the lower levels of cyanide, though still greatly above normal, that persisted for many days. Furthermore, Amdur's patients improved as blood thiocyanate rose or remained high.

—F. RIEDERS, Ph.D., and H. BRIEGER, M.D.
The Jefferson Medical College of Philadelphia

From reading Dr. Amdur's article it would seem that in Case 1 the patient died of cyanide intoxication and that in Cases 2 and 3 they were initially suffering from cyanide poisoning. Their later

symptoms as well as most of the symptoms in Cases 4, 5, 6, 7, and 9 could have been due to the presence of high levels of thiocyanate ion in their blood. The reasons that I give for this follow:

1. In Case 2, the patient was admitted with symptoms and signs which could have been due to excess of either cyanide and/or thiocyanate; however, after receiving treatment *specifically* indicated for cyanide intoxication, he made a remarkable recovery within 12 hours. At this time he was without symptoms, the blood level of thiocyanate was 16 mg %. This fell to 13 mg % over a period of five days. In Case 3 the patient similarly had symptoms which responded immediately to the same treatment. He continued then to have some symptoms which were suggestive of thiocyanate intoxication with blood levels to substantiate this conjecture.

2. I disagree on the basis of various literature references that blood levels of thiocyanate below 12 mg % are productive of symptoms. In fact, the recommended therapeutic dosages are designed to give blood levels from 8 to 12 mg/100 ml of blood. It is also said that symptoms of cerebral derangement do not appear until thiocyanate blood levels have exceeded 20 mg %.

3. Thiocyanate is distributed in the extracellular fluid space and eliminated *very* slowly. Very high levels of thiocyanate would be maintained for several days. With respect to the above-presented arguments, it might be noted that in Case 3 the symptoms *disappeared* while the thiocyanate level was increasing from 15 to 23 mg %. Needless to say, this is not what one would expect if the symptoms were due to thiocyanate.

I frankly do not know what to make of Case 8. I suspect that this does not actually belong with the others.

—CHARLES E. LEWIS, M.D.
The University of Kansas School of Medicine

I am very grateful for the opportunity to comment further on the material which you published in the JOURNAL OF OCCUPATIONAL MEDICINE for December, 1959. I am impressed by all that Dr. Brieger has set forth, particularly with regard to biotransformation and the likelihood that cyanide and not thiocyanate may be the villain in exposures to acetonitrile. Despite all of this, I find myself returning to my original premise after much the same reasoning as I did when I initially set my impressions down for publication.

I felt that, though biotransformation of thiocyanate to cyanide probably does occur, this is again a two-way reaction, and that reconversion to thiocyanate in equilibrium to the conversion to cyanide is probably the true situation. Since this was a clinical study and not an exercise in toxicity, I should have indicated perhaps that my therapeutic intent, particularly in Cases 1 and 2, was not the control of any concentration of cyanide ion in the blood, but rather the control of what was most obviously a reduction in the circulating blood volume. The sodium thiosulfate which was employed was somewhat in the nature of a salve to my own conscience, and consisted of only 10 cc of 10% in the first of each veniclysis flask for each patient. It was my impression that oxygen and the fluids restoring blood volume controlled such hypoxia as existed. I would have hesitated to use any of the specific nitrites recommended for cyanide therapy, because of the fear that further vasodilation would have increased, rather than lessened, the degree of shock which these people exhibited.

I have been suspicious somewhat in this entire exposure study, that a "red herring" stemming from one of the associated exposure materials—i.e., phenolic resin, aliphatic amine, or thiokol—may have been responsible for altering the host response to acetonitrile. Acetonitrile had been used without incident on many prior occasions, and in comparable work situations with or without these additives, until such time as the innovation of the application of heat to the solvent material and the reduction of air flow during its application precipitated disability. This would support the assertion of Dr. Brieger, with which I am entirely in accord, that the intact acetonitrile molecule possesses none or negligible intrinsic toxicity. The "red herring" may explain the response in Case 8, whose disability resembled chiefly a hepatic response, and may also explain somewhat the urinary complaint which appeared in Cases 2 and 3.

The persistent character of the complaints suggests to me the pharmacological behavior of the thiocyanite ion, i.e., vascular dilatation, as opposed to that of the cyanide ion, which would produce primarily hypoxia and possibly vasoconstriction. There does seem to be some lack of correlation between symptomatology and blood levels for cyanide and thiocyanate. This has been pointed out particularly by Dr. Lewis, who noted that in Case 3 the symptomatology lessened considerably although the serum thiocyanate level during the same initial 24-hour period rose from 15 to 23 mg %. This would seem to indicate that complaint could not be due to cyanide; however, it should be noted also from the reported findings, that the blood levels of the cyanide ion during the same initial period rose from 970 μg % to 1,088 μg %. As happens so many times, we are always being plagued by those things which we did not do, but which hindsight indicates we should have done. It might have been most illuminating had I secured thiocyanate in Case 1 (fatality).

—MARVIN L. AMDUR, M.D.
Buffalo Industrial Medical Center

Editorial

How Much Occupational Disease?

HOW MUCH occupational disease occurs in the United States? For the country as a whole, it honestly must be admitted that no one can make a defensible guess.

We have the beginning of an understanding of the number of cases that occur in California. Here a unique combination of circumstances exists that makes it possible to get some idea of a true number for this one state. These favorable circumstances are: (1) a system that brings in a Doctor's First Report of Work Injury from the practicing physicians throughout the state, (2) a group of capable and interested investigators of the problem in the Department of Public Health, and (3) support of such studies by the Department of Industrial Relations which administers the California Workmen's Compensation Act.

In our lead article in this issue, Berkov and her associates say that approximately 20,000 First Reports of occupational disease are received each year in California. These are cases which cause disability beyond the day of injury or require medical treatment, and occur among workers covered by the California Act. The involved workers are treated by some 4,000 doctors of medicine, or about one-fourth of those actively engaged in practice in the state. Additionally, an estimated 400 osteopaths and 100 chiropractors also submit reports of cases in a typical year.

Waiving some obvious objections for the moment, we make a conservative guess that the experience reported in California represents one-tenth of the experience for the nation; that annually in the United States there occur 200,000 cases of occupational disease labeled as such by the treating physicians; and that 45,000 physicians are involved in the provision of such treatments.

Johnstone has drawn attention[1] to the shortcomings of the Doctors' First Report as an indicator of occupational disease incidence in California. He doubts that many practitioners apply the rigorous procedures needed to support answers to three fundamental questions: What is the exact nature of the illness in the worker? What is the precise substance that is believed to have caused this illness? Were the circumstances of the particular worker's exposure such that the substance in question could have caused the illness? The average practitioner should be able to answer the first question on the basis of his general capacities as a clinician. But the second and third questions can be answered only from direct observation and inquiry at the place of work, and from information dug out of books and journals. The busy practitioner rarely has the time or taste for these efforts, or worse, does not even know that such efforts are necessary.

The study by Berkov and her associates does not meet the challenge presented by Johnstone (nor, in all fairness, was it so intended). The study compared Doctors' First Reports with what the reporting doctors themselves finally knew about their cases, and showed that the First Report satisfactorily represents this knowledge. In this sense, the First Reports are both *accurate* and *complete*. Unexplored in this study are *accuracy* and *completeness* at a different level, the reporting doctor's capacity to deal with the subject in the first place.

What is needed now, of course, is a second and more difficult study: careful investigation of a random sample of "reported" occupational disease cases to discover the "real" cases. As a minimum, each such case investigation should include an industrial hygiene study at the workplace and a second examination of the involved worker by a clinician who is a specialist in occupational disease diagnosis. The conclusions of such investigations would be matched with the Doctor's First Reports to determine the proportion of "false positives." The regularly produced California figures of incidence then with confidence could be extrapolated to the nation as a whole. But more importantly, the experience gained in the conduct of the studies could be applied by others who will wish to make comparable investigations elsewhere.

[1]Johnstone, R. T.: *Occupational Diseases: Diagnosis and Proper Method of Reporting. J.A.M.A.* 168:1844-1847 (Dec. 6) 1958.

Abstracts of Current Literature

Surgery

Practical Problems in the Treatment of Hepatic Trauma. A. J. McAllister,* M. F. Hicken, and R. P. Clark. Am. Surgeon 27:529 (July) 1961. (*Medical Arts Building, Salt Lake City, Utah)

Hepatic injuries are classified as penetrative, lacerative, and subcapsular. A general discussion of the cause and effects of the various types of injuries is presented. It is emphasized that subcapsular injuries to the liver can be quite severe and, if not adequately treated, lead to delayed ruptures or to erosion into the large bile ducts with evacuation of the contents into the duodenum causing hemobilia. It can be followed by severe hemorrhage. The author feels there is no place for conservatism in the treatment of liver injuries. Treatment must stop hemorrhage and extravasation of bile, debride nonviable liver fragments, provide adequate drainage for liver wounds, and prevent infection. It is stressed that packing is seldom of value. The treatment of choice is direct exposure of the injured liver with individual ligation of the vessels, debridement of necrotic tissue, and the placement of rubber drains into the depths of the liver wound. Experimental work was done with dogs by packing liver injuries with absorbable types of implanted material. In most instances, these substances were rejected by the liver, and, in a few where they remained in place, there was a marked foreign-body reaction. When one encounters a central hematoma with the capsule intact, this area of the liver should be opened, the necrotic tissue evacuated, and adequate drainage established. —A.A.B.

Restoration of Rheumatoid Finger Joint Function. A. E. Flatt.* J. Bone & Joint Surg. 43:753 (July) 1961. (*Department of Orthopedic Surgery, University Hospital, Iowa City, Iowa)

In an interim report on the use of a metallic articulated prosthesis for replacement of joints of the metacarpal-phalangeal or proximal interphalangeal joint of the rheumatoid hand, Flatt notes that, in the rheumatoid hand, contracture of the intrinsic mechanism produces the swan-neck finger deformity. Contracture of this mechanism has the effect of pulling the proximal phalanx in a proximal and palmar direction in relation to the metacarpal, eventually causing dislocation. He notes that the usual release operations in which the oblique fibers of the aponeurosis are sectioned are not always successful in overcoming the swan-neck deformity, and a more extensive release may be indicated. In addition to a swan-neck deformity of the proximal joint in the rheumatoid hand, commonly there will be additional deformities in the hand. The common deformity is ulnar displacement of the extensor tendon overlying the metacarpal-phalangeal joint with an associated synovitis of this joint with partial displacement of the phalanx ulnarward to eventual complete ulnar dislocation. For this, he describes an operative repair if the extensor mechanism is intact.

In addition to these two major surgical approaches to the rheumatoid hand, there is a place for a prosthesis to replace a hand joint. He feels that the indications for prosthetic replacement in an interphalangeal joint are (1) gross joint destruction, (2) persistent swan-neck deformity, (3) ankylosis of the joint in a nonfunctional position. The indications at the metacarpal joint are (1) gross joint destruction, (2) palmar dislocation of the joint, and (3) persistent ulnar drift with either joint destruction or palmar dislocation. To perform the operation at either joint, an intact extensor mechanism is necessary. A boutonnière deformity at the proximal interphalangeal joint is a contraindication to the use of the prosthesis, and the author has not had success with repair of this defect in the rheumatoid hand. The best approach to this deformity is fusion in the functional position. The technique of prosthetic insertion is described, and the results in 57 cases are presented. The author feels that surgery on the rheumatoid hand should be carried out in a quiescent period. —O.E.M.

Complete Dislocation and Subluxation of the Acromioclavicular Joint. M. A. Lazcano,* S. H. Anzel, and P. J. Kelley. J. Bone & Joint Surg. 43: 379 (April) 1961. (*Mayo Clinic, Rochester, Minn.)

The authors note that 35 conservative methods and 28 operative methods have been advanced for the treatment of dislocation of the acromioclavicular joint. The operative treatment consists of four types: (1) fixation of the acromioclavicular joint by wires, screws, etc., (2) arthroplasty with excision of the outer end of the clavicle, (3) fixation of the coracoid to the clavicle by a screw, and (4) repair of the coracoclavicular ligaments. Conservative therapy has been of three types: (1) adhesive dressing, (2) various harnesses and braces, or (3) various hanging-type casts. In the opinion of Urist, Stubbins, and McGaw, some type of cast, either spica or circular body, which exerts positive relocation pressure on the clavicle is best.

The present study concerns 43 patients with acromioclavicular dislocation, 40 having symptoms enough to warrant treatment. Eight patients had an excision of the distal inch of the clavicle. In seven more, this was combined with an additional procedure, usually transfixion of the clavicle to the coracoid with a lag screw. In only one of these cases was there residual limitation of motion and mild pain. In 14 cases, a variety of fixation devices (screws, wires, etc.) was used for fixation of the acromioclavicular joint. In this group, seven patients had an excellent result; three had fair results; and three had poor results.

Abstracts

Seven patients were treated by fixation of the clavicle to the coracoid with a lag screw. The authors report this is a tedious operative procedure. Four patients had excellent results, and three had fair results. After review of their cases, the authors felt that more consideration should be given to a closed method as suggested by Urist. If a normal range of painless motion is not achieved by this method, then simple excision of the distal end of the clavicle could be done at a later date. —O.E.M.

Noise and Hearing

The Effects of Ionizing Radiation on the Ear. S. Borsanyi,* C. L. Blanchard, and B. Thorne. Ann. Otol. Rhin. & Laryng. 70:255 (March) 1961. (*University of Maryland, Baltimore, Md.)

Radioactive treatment for head and neck cancer often may incidentally involve the cochlea. These authors studied the effect of from 4,000 to 6,000 r of radiation in the neighborhood of the cochlea on the hearing of 10 patients. Except for a slight (and apparently not statistically significant) transient conductive threshold shift, no effects were observed. —W.D.W.

Prediction of Speech Hearing Loss for Older Industrial Workers. T. Farrimond.* Gerontologia 5: 65 1961. (*Medical Research Council, Liverpool, England)

Compensation for hearing loss ideally depends on the degree to which the loss reduces the ability to perceive ordinary speech. This reduction, however, is usually not measured directly but is rather calculated from the pure-tone losses at 500, 1000, and 2000 cps, using empirical equations validated on young men using conventional phonetically balanced lists of single words. In this study, the author uses a measure of speech perception more nearly like the actual social situation, i.e., a set of everyday sentences. Pure-tone thresholds are related to the ability to understand these sentences for 70 young (40 and younger) and 110 older (older than 40) workers. It is found that a slightly better prediction of speech perception is obtained if one uses the threshold at 250 cps in addition to the ones cited or perhaps instead of 2000 cps. Although the author does not make the point specifically, the data show that there were no differences between the two age groups: predictions of loss of ability to understand speech, using an equation based on pure-tone thresholds, are just as valid for older workers as for younger. —W.D.W.

Middle Ear Muscle Reflexes in Man. I. Klockhoff.* Acta oto-laryng. Supp. 164, 1961. (*Karolinska Sjukhuset, Stockholm, Sweden)

In this monograph, the author describes extensive and ingenious studies of the protective middle-ear reflexes using an apparatus, devised by Anderson, which allows stapedius and tensor activity to be inferred from changes in the "relative reflection of a (550-cps) probe tone transmitted into the closed auditory canal." Since auditory stimuli (loud sounds) result in contraction of both muscles, techniques were developed to study each independently. It is demonstrated that cutaneous electrical stimulation (1-msec pulses of 1 to 4 volts at 50 pulses per second) in the external auditory canal arouses, for a second or two, only the stapedius of the ear concerned, although the maximum strength of action that could be elicited in this way was always less than the maximum produced by acoustic stimulation. Similarly, contraction of the tensor tympani, without involvement of the stapedius, is effected by directing a strong puff of air toward the eyes; apparently this action of the tensor is part of a general startle reaction. He suggests that the normal tensor tympani reflex is probably myotatic in nature, i.e., that it contracts when stretched (the stretching ordinarily being caused by contraction of the stapedius). The practical application of these tests in the differential diagnosis of hearing losses is illustrated. For example, when no stapedius reflex can be elicited, then unless the stapedius is paralyzed by facial palsy a conductive type of loss is indicated. In such a case, the locus of the conductive impairment can be deduced from the nature of the tensor tympani response. If the tensor tympani responds, though weakly, then the trouble is probably otosclerotic fixation of the stapes, with no other complications. On the other hand, if no response can be obtained, then other lesions of the middle ear are probably present, impeding the mobility of the eardrum. Finally, if the tensor response is much larger than normal, a physical interruption of the ossicular chain is indicated. —W.D.W.

Auditory Adaptation. J. F. Jerger.* J. Acoust. Soc. America 29:357 (March) 1957. (*Northwestern University, Evanston, Ill.)

The decline in the ear's response under sustained stimulation, a phenomenon variously labled "auditory fatigue," "perstimulatory fatigue," "auditory adaptation," was measured for seven frequencies from 125 to 8000 cps and seven intensities from 30 to 90 db using the median-plane-localization method. The amount of adaptation at each one-minute interval during the five-minute period of sustained stimulation was computed by substracting the SPL (sound-pressure level) required on the control ear for a match of each interval from the mean of the three SPL's required on the control ear in the three matches made prior to stimulation. The mean adaptation curve for each experimental curve was derived by averaging the individual curves of the 10 experimental subjects. Results indicated that high frequencies show considerably more adaptation than low frequencies. Adaptation after a five-minute stimulation at 125 cps is quite negligible even at an SPL of 90 db. At 4,000 cps, however, a

substantial amount of adaptation has occurred after only one minute of sustained stimulation at levels of 50 db and above. There appears to be a fairly linear relationship between adaptation, in decibels, and sensation level, in decibels, in the range from 10 to 80 db for all frequencies. Results indicate that the duration at which maximum adaptation occurs appears to be a function of both frequency and intensity. A maximum is reached after three minutes for 125, 250, and 500 cps, but from 1000 to 8000 cps only the curves for the very low intensities show any tendency to stabilize at a maximum value. At higher intensities, the curves continue to drop even after the fifth minute. There is also a great deal of variability of adaptation between subjects. One subject showed adaptation of 5 db after the fifth minute of sustained stimulation, while another showed a decline of 45 db. —R.G.H.

Environmental Physiology

A Physiologic Criterion for the Determination of Environmental Limits for Everyday Industrial Work. A. R. Lind.* Proc. 13th Internat. Cong. Occup. Health, p. 882, 1960. (*Department of Human Anatomy, University of Oxford, England)

The classic studies of Nielsen (Sand. Arch. Physiol. 79: 193, 1938) demonstrated that the rise in deep body temperature during work at a constant rate is the same in climates ranging from comfortably cool to moderately warm. Under these conditions, therefore, the rise of body temperature does not indicate a strain on thermoregulatory processes but is in fact a regulated rise. Lind confirms Nielsen's findings but extends the range of environmental temperatures investigated upward to levels at which rectal temperature increased with each increment in environmental temperature. The author regards the level of environmental heat at which transition from a constant to a progressive rise in rectal temperature as a useful criterion for the upper permissible limit of heat conditions for industrial work. The "neutral" zone in which work can be conducted without excessive strain will vary with age, sex, acclimatization, clothing, and work rate. In the subject of this report, who walked without clothing on a treadmill at 5.5 km/hr up a 5% slope, the neutral zone extended from 57.4° to 82.4°F (13° to 28°C) Effective Temperature. —D.M.

Ermüdung und Erholung im Arbeitsablauf (Fatigue and Recovery During the Course of Work). G. Lehmann.* Proc. 13th Internat. Cong. Occup. Health, p. 878, 1960. (*Max Planck Institute für Arbeitsphysiologie, Dortmund, Germany)

Fatigue, from the viewpoint of the physiologist, is practically always caused by an oxygen supply which is less than the demand. If waste products accumulate during steady work, fatigue should increase linearly with time. Fatigue, however, increases exponentially. One might speculate that this exponential progress is caused by some "fatigue metabolite" which in turn inhibits metabolism. Recovery also has been found to progress exponentially. One must conclude, therefore, that short phases of work interrupted by short intervals of relaxation would result in less fatigue than one long rest period interposed between several hours of steady work. The least fatigue results from alternating contraction and relaxation in phases of 1.2 seconds for the longest and 0.3 seconds for the shortest muscles. If the phases become too slow (i.e., static work) or if they become too fast (and the load per unit cross section increases), either the oxygen supply or the recovery periods will become inadequate. If the work rhythm cannot be made optimal, rest periods should be interposed. They might be short enough not to appear as an interruption of the work process.

The workers must be trained to understand this principle and to cooperate by self-observation and trial. Coordination of work and rest periods is essential not only for physically hard-working men but also for men doing so-called light work, which mainly involves the small muscles. In this case, the load per unit area of muscle cross section might be high and cause local fatigue, which the worker will attempt to overcome by will power, thus leading to mental fatigue. Mental fatigue, however, usually should not occur from the unphysiological use of muscles but is experienced chiefly from conscious and productive mental activity. —D.M.

Ermüdungsbekämpfung in der Automatisierten Industrie (Fighting Fatigue in Automated Industry). H. Schmidtke.* Proc. 13th Internat. Cong. Occup. Health, p. 921, 1960. (*Max Planck Institute für Arbeitsphysiologie, Dortmund, Germany)

Past attempts at quantitatively analyzing tension fatigue have been unsuccessful. Motivation was assumed, without justification, to have been optimal. This often is not true; and lack of motivation may have a greater effect on efficiency than actual fatigue, especially in supervisory or control jobs with little physical or mental activity. Tension fatigue was always measured in relation to work duration, and thus these studies resulted merely in an estimate of the time required to cause a certain degree of fatigue. This one variable, *time*, does not alone determine the degree of fatigue, and therefore such measurements cannot solve this complex problem.

The author introduces other variables by studying the nature of work and the efficiency of performance in relation to the duration of work and rest periods. Industrial jobs may be classified in three groups: (a) work requiring continued attention and readiness for psychomotor reaction over a wide observation field (such as a steel worker at a rolling press); (b) work requiring continued attention of few signals (such as a radar observer in aviation control); and (c) work requiring continued attention with hardly any

activity or change of signal (such as a watchman at a signal panel of a power plant).

Using an eight and one-half hour period with task situations simulating the three types of work, the author studied the effects of varying periods of work and rest. For example, in a task requiring continued concentration on one object, the optimum relation between work and rest periods is 7.3:1, the rest periods being more effective when short and frequent rather than of long duration and less frequent, as in Lehmann's studies of physical fatigue. Moreover, monotony is an important factor leading to loss of attention. Only genuine rest with as little individual isolation as possible at the place of work will reduce monotony. —D.M.

Experimental Toxicology

Acquired Porphyria in Man and Rat Due to Hexachlorobenzene Intoxication. R. K. Ockner* and R. Schmid. Nature 189:499 (Feb. 11) 1961. (*Boston City Hospital and Harvard Medical School)

Rats were fed a diet containing 0.2% hexachlorobenzene; urinary and fecal excretion of porphyrins and porphyrin precursors were determined periodically. After the rats were killed, tissue porphyrins and liver catalase were measured. Within one month, 13/33 rats died, exhibiting terminal tremor, ataxia, weakness, and paralysis but no evidence of porphyria. The remaining 20 showed significant increases in porphyrin and precursor excretion after two to eight weeks. When hexachlorobenzene administration was discontinued after peak excretory levels were reached, the porphyrin metabolism disturbance was rapidly reversed. An irreversible porphyria resulted if high levels of excretion by continued feeding were maintained, and the rats died with neurological complications. The results demonstrate that chronic ingestion of hexachlorobenzene by the rat may result in porphyria, heretofore assumed to be a genetic disease in man. Recent outbreaks of cutaneous porphyria in Turkey may have resulted from repeated exposures to hexachlorobenzene. —R.E.E.

Toxic Hazard from Formulating the Insecticide Dimethoate in Methyl "Cellosolve." J. E. Casida* and D. M. Sanderson. Nature 189:507 (Feb. 11) 1961. (*University of Wisconsin and Chesterford Park Research Station, Essex, England)

A fraction was separated during the purification of commercial dimethoate which potentiated the rat oral toxicity of the pure compound. The potentiator, perhaps a salt of O-methyl-S-(N-methylcarbamoylmethyl)phosphorothioic acid or a closely related compound, was active in the rat by oral and intraperitoneal routes but not by skin absorption. Technical dimethoate in methyl Cellosolve, which had been in storage in England for seven months, decreased the original rat oral LD_{50} from 150-250 mg/kg to 30-40 mg/kg; after nine months storage under tropical conditions the LD_{50} was < 15 mg/kg, and after storage at 100°C for sixty-three hours the LD_{50} was 8 mg/kg. Mammalian toxicity increased seven fold before insecticidal activity was altered. The most toxic fraction of the decomposed dimethoate-methyl Cellosolve formulation was an O, O-dialkyl-S-N(N-methylcarbamoylmethyl)phosphorothiolate with one or two methyl groups of dimethoate replaced by 2-methoxyethyl groups; its rat oral and intraperitoneal LD_{50} values are about 1 and 0.5 mg/kg, respectively. Of several monohydroxylic solvents tested, only methyl and ethyl Cellosolve formulations demonstrated increased toxicity after short-term storage at 70°C; polyhydroxylic solvents did not display this property. Several other organic phosphate insecticides also increased in toxicity after reaction with methyl Cellosolve at 70°C for eight days. —R.E.E.

The Acute Inhalation Toxicity of Heated Aerosols of Three Hydraulic Fluids. L. Feinsilver,* and others. Am. Indust. Hyg. A. J. 22:14 (Feb.) 1961. (*Army Chemical Center, Maryland)

Rats and dogs were exposed four hours to both unpyrolyzed aerosols and aerosols pyrolyzed at 520°, 1,020°, and 1,560°F of the following fluids: OS-40, a phosphate ester; XF-408, a mixture of a silicone oil and a fluorochlorohydrocarbon; and MLO-5277, an organosilicate. All aerosols were eye and respiratory irritants, and toxicity was generally proportional to the nominal concentration of the aerosol (0.3-4.4 mg/liter) and the temperature to which the aerosol was subjected. The one exception was XF-408, which caused acute tubular nephrosis in dogs exposed to the unheated aerosol and to the aerosol heated to 520°F but not when heated to 1,020° or 1,560°F (although the incidence of deaths was greater in the 1,560°F tests). OS-40 produced severe pathologic changes in rats and dogs only when the nominal concentration of the aerosol heated to 1,560°F was 3.5-4.3 mg/liter. It was the least toxic of the three fluids tested. MLO-5277 was considered to be the most toxic, since animals died at the lowest concentrations of 0.51 mg/liter of the undecomposed aerosol and at low concentrations of the pyrolzed aerosols. —R.E.E.

Tumour-Promotion by Lime Oil in the Mouse Forestomach. W. E. H. Peirce.* Nature 189:497 (Feb. 11) 1961. (*London Hospital Medical College, London, England)

Data are presented from studies to determine the tumor-promoting activity of lime oil. Albino mice were dosed singly by stomach tube with 9,10-dimethyl-1,2-benzanthracene (DMBA) or with 3,4-benzpyrene (BP), in polyethylene glycol (PEG), followed in three weeks with 40 weekly doses of 0.05 ml undiluted lime oil. Other groups of animals received the initiators, or PEG, or lime oil only. Forestomach tumors were seen in all animals treated with initiators except one group given 50 μg BP, but more tumors developed in the cor-

responding groups dosed in addition with lime oil. The difference between the two groups in number of tumors induced was more apparent when only tumors > 3mm in diameter were considered. No tumors developed in untreated animals; 2 in 8 were seen in the group given only lime oil, and 3 in 11 in the group given PEG. Only one malignant tumor was found, a squamous-cell carcinoma in an animal treated with 200 μg BP and lime oil. Although both the hydrocarbons and lime oil are carcinogenic to an extent, their combined activity appears more than additive. No tumors were seen in the glandular part of the stomach or elsewhere in the alimentary canal.

—R.E.E.

Internal Medicine

Effect of Alpha Particle Hypophysectomy on Disseminated Cancer of the Male Breast. J. D. Constable,* J. H. Lawrence, J. L. Born, C. A. Tobias, P. E. Ariotti, F. F. Sangalli, R. C. Carlson, and P. Toch. J.A.M.A. 174:1720 (Nov. 26) 1960. (*Donner Pavielion and Lawrence Radiation Laboratory, University of California, Berkeley, Calif.)

This is a preliminary report by the authors, who have been interested in particulate radiation generated by the cyclotron; such radiation has great penetration with little scatter. For six years, they have used this type of radiation to depress pituitary function in cases of disseminated breast cancer.

Various methods have been used in the treatment of disseminated breast cancer in the male: orchiectomy, surgical hypophysectomy, and adrenalectomy. It has been apparent in male patients that androgens, before and after castration, aggravate the condition, while in the female the response is good. Estrogen therapy in the male has not proved satisfactory; it may be palliative but frequently aggravates the disease. At present, orchiectomy seems to be the procedure of choice in inoperable male breast cancers or in metastatic or recurrent disease.

The authors present two cases. In the first, death occurred before any results of pituitary radiation could have been expected. In the second, x-ray improvement in the bony metastases was noted six months after alpha-particle radiation to the pituitary; in addition to metastatic improvement, there was also excellent clinical improvement.

The authors conclude that male breast cancer, if it is hormone-dependent, depends on testicular androgens or on androgen-like adrenal steroids. They speculate that pituitary ablation may result in remission. —S.H.S.

Iatrogenic Jaundice. F. Schaffner.* J.A.M.A. 174: 1690 (Nov. 26) 1960. (*11 East 100th St., New York, N. Y.)

The liver is a frequent target of toxic drug action. The purpose of this study was to consider drug reactions from the clinical viewpoint. The author first discusses poisons with predictable reactions such as carbon tetrachloride. Clinical types of iatrogenic jaundice are then discussed.

It is pointed out that the clinical features are not distinctive; all patients have prodromata such as malaise, anorexia, fever, and some have a distaste for tobacco. The combination of jaundice, dermatitis, or purpura, however, is highly suggestive of a drug reaction.

Laboratory tests separate drug-induced jaundice into three groups: (1) hemolysis, with indirect bilirubinemia and no bilirubinuria; (2) hepatocellular damage, with positive flocculation tests and high serum transaminase; and (3) cholestasis, with high serum alkaline phosphatase. The mechanism in cholestasis is intrahepatic obstructive jaundice, now more frequent than any type of drug jaundice. Sulfonamides, phenothiazines, diuretics, and antidiabetic drugs are the chief offenders. Hepatocellular damage is caused by such drugs as cinchophen, iproniazid, pyrazinamide, zoxazolamine, metahexamide, and sulfamethoxypyridazine. Iproniazid as an incidence of toxicity reactions of less than 0.1% but a mortality from 20% to 50%. Hemolytic jaundice can occur during therapy with acetanilid, acetophenetidin (Phenacetin), sulfonamides, glucosulfone (Promin), methylphenylethyl hydantoin (Mesantoin), paraaminosalicylic acid, and quinine.

The author discusses therapy and prevention. When using drugs known to produce jaundice, careful follow-up and periodic determinations of the serum transaminase and serum alkaline phosphatase are mandatory. —S.H.S.

Medical Care Administration

The Battle for Your Health Dollar. R. H. Berg.* Look, April 11, 1961, p. 24. (*Look Magazine, Madison Ave., New York, N.Y.)

Prepaid medical care is here to stay. The big question today is how is it to be paid for and shall it be through Blue Cross-Blue Shield, the commercial insurance companies, or the federal government. The decision Congress makes on the "over 65" may determine whether medical care for everyone will be tied to social security.

The author points out that, while 72% of all Americans have health insurance, these policies only pay 24% of the nation's medical care bills. Many medical care costs are not considered insurable. Blue Cross has 78 separate plans, each writing its own policies; the 68 Blue Shield plans vary just as widely.

Commercial insurance can compete with the "Blues" because they use an experience-rating system to establish premiums and benefits, adjusting these up or down depending on whether the group represents a high or low risk. In some areas, they have been accused of siphoning off the low-risk age groups so that the "Blues" have been forced to continue to increase their rates.

Studies by the Department of Health, Educa-

tion, and Welfare in 1959 showed that the administrative costs would be less under social security than through Blue Cross or commercial insurance.

Two of the leaders in the hospital and medical care field expressed surprise that the social security mechanism should be opposed by the American Hospital Association and that AHA should be backed by the American Medical Association in its condemnation of the plan. It was pointed out that hospitals are overwhelmingly influenced by the physicians, and while they are workshops for the physicians they are places for patients. Hospital people should work for the best interest of the people.

The way the problem is settled for persons over 65 may set the pattern for cost and quality of medical care for all of us. —A.B.

A Doctor Looks at Cost of Care. B. W. Rawles, Jr.* Trustee 14:28 (July) 1961. (*Richmond Memorial Hospital, Richmond, Va.)

Except in the admission of extreme emergencies, the physician controls a large part of the cost of hospitalization. It is the physician who decides whether hospitalization is necessary, and he is responsible for ordering drugs, laboratory procedures, radiological examinations, physical therapy, electrocardiograms, and other special examinations and treatments. It is his decision when to discharge the patient.

The dollar waste of unnecessary hospitalization and excessive utilization is of growing concern to Blue Cross, to private insurance, and to the public in general. A continued increase in the cost of food supplies and labor over which the physician or hospital has no control is going to bring about progressive increases in prepayment rates. We should not add to it the cost of unjustified hospitalization.

If patients are unnecessarily admitted, hospital beds may not be available for patients who are actually ill. In many hospitals, bed reserves are almost nonexistent because the beds are occupied by patients who might just as well be treated as outpatients or treated at home.

The physician should welcome an opportunity of self-analysis of his medical practice patterns, to the end that the quality of the care rendered to his patients will be improved and the cost kept to a minimum, without endangering the medical service rendered the individual. —A.B.

Merging the Three Images of the Doctor: His Own, the Community's, the Ideal. F. Lucchesi.* Trustee 14:7 (April) 1961. (*Albert Einstein Medical Center, Philadelphia, Pa.)

People believe the physician of today is not the self-sacrificing, devoted practitioner and family counselor he formerly was. The physicians would be deceiving themselves if they refused to admit that the external image of organized medicine, justly or unjustly, is somewhat unfavorable. A few greedy doctors may give the impression that the fees of all physicians are unjustifiably high. American medicine has been so preoccupied with forestalling socialized medicine that they may have failed to recognize the many roads which can lead to socialization.

Hospitals, health and welfare councils, Blue Cross, labor unions, and even governmental agencies have developed certain misapprehensions concerning doctors. Among paramedical professional groups the feeling is that the medical profession has been aloof and uninterested in their efforts to serve the public.

The profession must improve its relation with those agencies that are also trying to serve the health of the community. They must understand what these agencies are trying to do and some of the problems they face. All needs of the sick in the community must be the concern of the medical profession.

The author suggests that physicians earnestly endeavor to merge their internal and external images—what physicians think of themselves and what the public thinks of them—into a new and true image, to give the public the true image of the physician of the 1960's. —A.B.

Radiation Health

Radiation Injuries and Statistics: The Need for a New Approach to Injury Litigation. S. D. Estep.* Michigan Law Rev. 59:259 (Dec.) 1960. (*University of Michigan, Ann Arbor)

In the field of compensation for radiation injuries, the bar and the legislatures have not kept pace with other specialties involved. Current litigation procedures are not able to cope with many potential radiation injuries, particularly where the injury is latent and is nonspecific. Under existing rules, the burden is on the plaintiff to show that the relationship is "reasonably certain" or "reasonably probable," which can be interpreted to mean "more probable than not." The result is a "lottery" type of justice.

A "contingent injury fund" administered either by the private insurance industry or by government is one possible solution to the problem. Under this system, defendants contribute to a fund in proportion to the increased liability caused in terms of radiation-exposure dose above the maximum permissible. The liability is then discharged. The natural incidence of each nonspecific disease would be covered either by donations from the defendants or from all exposed workers. To recover compensation from the fund, a victim merely needs to prove that he has the particular disease. Some quantitative evaluation can be made now with respect to the compensation for leukemia. The radiation-induced incidences for other latent and nonspecific diseases are less well known.

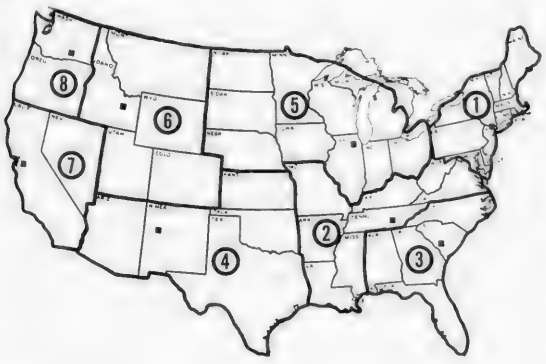
The complex skills required for this type of litigation probably exceed the technical competence of the existing system of lay juries,

judges, or tribunals. A panel of experts, consisting of biological and physical scientists and lawyers, could make a better estimate of the best present scientific judgment *for legal purposes.* —T.S.E.

Organization of a Radiation Hazard Emergency Response Capability. A. J. Breslin,* W. B. Harris and A. A. Weintraub. Am. Indust. Hyg. 22:59 (Feb.) 1961. (*U. S. Atomic Energy Commission, Health and Safety Laboratory, New York, N. Y.)

An AEC and Department of Defense Radiological Assistance Plan was inaugurated in 1958 to provide emergency technical assistance to state and local officials in the event that an accident with public health significance occurs during the utilization, handling, or transportation of radioactive materials. This assistance can be mobilized anywhere in the United States or its possessions by contacting one of eight AEC Operations Offices assigned a geographical region responsibility. The New York Operations Office emergency team organization is described. The teams are highly mobile and equipped to make hazard evaluations, which are provided to local authorities as soon as possible. The AEC has issued emergency recommendations to local civil authorities to prevent personal injury and property damage until additional technical assistance is obtained. The success of this assistance plan depends on wide dissemination of information regarding the availability of this AEC capability, the recognition by local authorities that radioactive materials are involved in an accident, and the application of the recommended emergency precautionary measures. —G.L.V.

EDITOR'S NOTE: AEC Radiological Assistance Capability for the rest of the country is available as indicated below:



1. New York Operations Office, 376 Hudson St., New York 14, N.Y.; phone: YUkon 9-1000.
2. Oak Ridge Operations Office, P.O. Box E, Oak Ridge, Tenn.; phone: Oak Ridge 5-7486, ext. 7607; or Oak Ridge 5-8611, ext. 7607.
3. Savannah River Operations Office, P.O. Box A, Aiken, S.C.; phone: MIdway 9-6211, ext. 3333 (through Aiken, S.C.), or PArk 4-6311, ext. 3333 (through Augusta, Ga.).
4. Albuquerque Operations Office, P.O. Box 5400, Albuquerque, N. Mex.; phone: ALpine 6-4411, ext. 38267.
5. Chicago Operations Office, 9800 South Cass Ave., Argonne, Ill.; phone: CLearwater 7-7711, ext. 2111 or 541.
6. Idaho Operations Office, P.O. Box 2108, Idaho Falls, Idaho; phone: JAckson 2-6640.
7. San Francisco Operations Office, 2111 Bancroft Way, Berkeley, Calif.; phone: THornwall 1-5620.
8. Hanford Operations Office, P.O. Box 550, Richland, Wash.; phone: WHitehall 2-1111, ext. 6-5441.

Abstracts

Compartmentalized Phantoms for the Standard Man, Adolescent and Child. R. L. Hayes* and M. Brucer. Internat. J. Appl. Radiat. 9:113 (Dec.) 1960. (*Medical Division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn.)

Radiation dosimetry phantoms for the 3-year-old, 12-year-old, and adult have been constructed of a series of plastic rectangular parallelepiped tanks based on anthropomorphic measurements. The phantoms have been useful in depth dose and energy absorption studies and neutron activation accident simulation. —T.S.E.

Industrial Hygiene Controls

Better Operation of Dust Control Systems. K. J. Caplan.* Arch. Environ. Health 2:248 (March) 1961. (*Uranium Division, Mallinckrodt Chemical Works, Weldon Springs, Mo.)

"If a company expends its capital for a dust control system, it has every right to receive a return on that investment." This introductory statement sums up the philosophy of this discussion, which covers a number of timely and pertinent observations concerning the lack of understanding of the basic requirements of dust control systems which may be installed to control a health hazard rather than directly increase production. Industrial management, however, must be willing to spend an amount of effort to secure proper maintenance of dust control systems. All specific recommendations and helpful hints are doomed to failure if management is not willing to make this minimal expenditure.

The author then discusses, in some detail, salient factors in the following categories: design for low maintenance; preoperational testing; education of users; and maintenance. Under each of these, he brings up some specific and important aspects of any control system which many individuals and corporations have been prone to ignore, since they are not included in the direct cost of production.

In conclusion, the author names six steps which he feels are necessary for better operation of

dust control systems. First, management must decide what it is going to obtain in return from the capital investment made in the dust control system. The second and third refer to design factors for minimum maintenance and ease of inspection; fourth, the necessity for testing and correction of defects before a system is put into operation; and fifth, the most important and most frequently neglected step, the education of the users and maintainers. After indicating the necessity for an operation and maintenance program for the system, the author states that he believes there will be enthusiastic acceptance by production supervision. The sixth and final step recommends the provision of adequate preventive maintenance inspection. —E.C.R.

A Progress Report on In-Plant Hazard Identification Systems. J. J. Duggan.* Arch. Environ. Health 2:269 (March) 1961. (*Union Carbide Corp., New York, N. Y.)

In this article, the author reviews the current status of a technique for communicating industrial hygiene and safety information to the persons who actually come in contact with the toxic materials. At the time this work was started, at least 15 different classification systems were in existence and used to varying degrees. One of the purposes of the committee listed below was to classify and simplify these systems. The committee was made up of representatives from the Bureau of Explosives, National Board of Fire Underwriters, Insurance Engineers, Fire Marshals' Association, U. S. Bureau of Mines, and several individual industries. In the proposed code, use was made of color, numbers, and shape or location to designate specific property or degrees. In spelling out the health hazard, the code recognized four degrees of hazard: low, minor, submajor, and major. Similarly, with fire and stability, 4 to 5 degrees of hazard are recognized. —E.C.R.

Plasma Jet. H. W. Speicher.* Arch. Environ. Health 2:278 (March) 1961. (*Westinghouse Electric Corp., East Pittsburgh, Pa.)

In this brief (one-page) article, Speicher calls attention to several environmental hazards which may be associated with the use of the so-called plasma jet to produce unusually high temperatures for use in research and development. In this application, the plasma jet is applied to an arc-gas device capable of heating gas to extremely high temperatures without combustion. By adding electrical energy continuously, the device can develop gas-flame temperatures in the range of 30,000°F.

Hazards mentioned in connection with this use of the plasma jet include noise which may require special facilities or personal protection for the operator; and eye protection, usually requiring a No. 6 shade welding plate mounted in a cover-all goggle or welding helmet. In addition to intense visible light, some gases, such as argon, may produce ultraviolet radiation of sufficient intensity to produce irritation of the exposed skin of nearby persons. Ventilation may be required to remove fine particulate material from the breathing zone of the operator and adjacent personnel. Unless ventilation is provided, oxides of nitrogen may exceed the allowable limit of 5 ppm. —E.C.R.

Dermatology

Isolation of Wart Virus in Tissue Culture. C. G. Mendelson* and A. M. Kligman. Arch. Dermat. 83:559 (April) 1961. (*Department of Dermatology, University of Pennsylvania, School of Medicine, Philadelphia, Pa.)

For more than 50 years warts have been known to be caused by an infectious agent. Now the agent has been grown in monkey kidney tissue culture and reinoculated into humans after six serial passages. Within a year, half of 20 subjects developed warts that were clinically and histologically typical. —W.L.E.

Immunologic Reactions to Penicillin. J. Harris* and J. H. Vaughn. J. Allergy 32:119 (March-April) 1961. (*University of Rochester, School of Medicine, Rochester, N. Y.)

Because use of skin tests alone does not regularly reveal persons sensitive to penicillin, these excellent workers explored the combined use of skin testing and hemagglutinating procedures. Of 13 persons with a diagnosis of probable penicillin reaction, 12 gave positive responses to either or both tests. Neither test alone was particularly informative. The difficulty with this work is that it tells us merely who has had a reaction, not who is going to develop one on the next exposure to penicillin.

As a side issue, the authors point out that patients with lupus erythematosus tend to have hemagglutinating antibodies to penicillin whether or not they have a past history of penicillin reactions. A search for penicillin antibodies may have diagnostic value in suspected cases of lupus erythematosus. —W.L.E.

Löffler's Syndrome Associated with Nickel Sensitivity. F. W. Sunderman* and F. W. Sunderman, Jr. Arch. Int. Med. 107:405 (March) 1961. (*Jefferson Medical College, Philadelphia, Pa.)

An interesting case is reported of a chemical engineer who developed chronic asthma following intense exposure to nickel carbonyl several years ago. Recent exposure to nickel carbonyl precipitated more severe asthmatic symptoms which required hospitalization. Chest films and eosinophil counts supported a diagnosis of Löffler's syndrome. At the same time, he had a chronic dermatitis of the hands; patch tests with nickel gave positive reactions. All lesions and symptoms cleared with rest in a mountain retreat. —W.L.E.

About the Authors

► BETH M. BERKOV holds a B.A. degree from the University of Wisconsin. From 1941-1946, she worked in the field of public opinion survey research. Since 1947, she has been a public health analyst with the California Department of Public Health, associated with various programs of that department, including the California Tumor Registry, the California Crippled Children Services Program, and for the past three years the Bureau of Occupational Health. She is an author of the recent publication "The Health of California Workers."

Mrs. Berkov is a member of the American Public Health Association. She is senior author of the article in this issue, "Evaluation of Occupational Disease Reporting in California." Associated with her as co-authors are the following persons:

GOLDY D. KLEINMANN, who received a B.A. degree from Hunter College and her M.A. degree from Columbia University in New York. She worked as a public health analyst with the California Department of Public Health from 1947-1952 and again from 1955 to the present. For the past six years, she has been associated with the Bureau of Occupational Health. Her main field of interest is occupational disease reporting. She is responsible for a number of departmental publications concerning occupational disease in California, including the annual series "Reports of Occupational Disease Attributed to Pesticides and Other Agricultural Chemicals."

JEAN E. POWERS, who attended Stanford University and University of California. She received her A.B. degree in Public Administration from the University of California. After graduation, she was employed as an economist for the Office of Price Administration and for the War Assets Administration. After World War II, she was employed as a statistician for the Firemen's Fund Insurance Group. She entered the California state service in 1950 and since that date has been in charge of the Work Injury Statistics Section of the Division of Labor Statistics and Research of the State Department of Industrial Relations. Under her direction, the division issues a monthly publication, "Work Injuries in California," an annual summary, "California Work Injuries," and special reports in the field of work injuries and workmen's compensation. She is a member of the American Statistical Association.

VICTORIA M. TRASKO, who is Program Adviser with the Division of Occupational Health, U.S. Public Health Service, at Washington, D.C. She is widely known for her work in occupational health and is the author of several publications dealing with occupational disease statistics and problems of reporting. Miss Trasko received her A.B. degree from Wilson College, Chambersburg, Pa. She has been with the Division of Occupational Health since 1938, working with state and local agencies on various program matters. For a six-month period in 1951-1952, she was on an industrial hygiene assignment with the Institute of Inter-American Affairs (now International Co-operative Administration) in Peru. She is a fellow of the American Public Health Association and a member of the American Industrial Hygiene Association.

► THEODORE KURZE, M.D., was recently appointed to the Salergni Collegium Chair of Neurological Surgery at the University of Southern California School of Medicine, Los Angeles, Calif. He is author of the article in this issue, "Practical Neurosurgical Principles in the Management of Multiple Injuries," presented as a paper in the session on Treatment of the Patient with Multiple Injuries by members of the surgery faculty of the University of Southern California at the Los Angeles Meeting of IMA.

Dr. Kurze received the M.D. degree from Long Island College of Medicine, Brooklyn, N.Y., in 1947. After a rotating internship at St. Monica Hospital, Phoenix, Ariz., he served residencies in neurosurgery at the V.A. Wadsworth Hospital and at Los Angeles County Hos-

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About the Authors

pital in Los Angeles. He served on the faculty of the University of California at Los Angeles for a year and then joined the faculty of the University of Southern California in 1956. He became head of the neurosurgery service at Los Angeles General Hospital early in 1961. He is certified by the American Board of Neurological Surgery. His research has centered on hypothermia in cerebral injury and on anaerobic cerebral metabolism.

► C. ANTHONY D'ALONZO, M.D., is co-author of the article in this issue, "Blood Pressure, Body Weight, Serum Cholesterol, and Smoking Habits Among Executives and Nonexecutives." He is Assistant Medical Director, E. I. du Pont de Nemours & Company, Wilmington, Del., and is responsible for the industrial medical services in which the data for the present article have their origin.

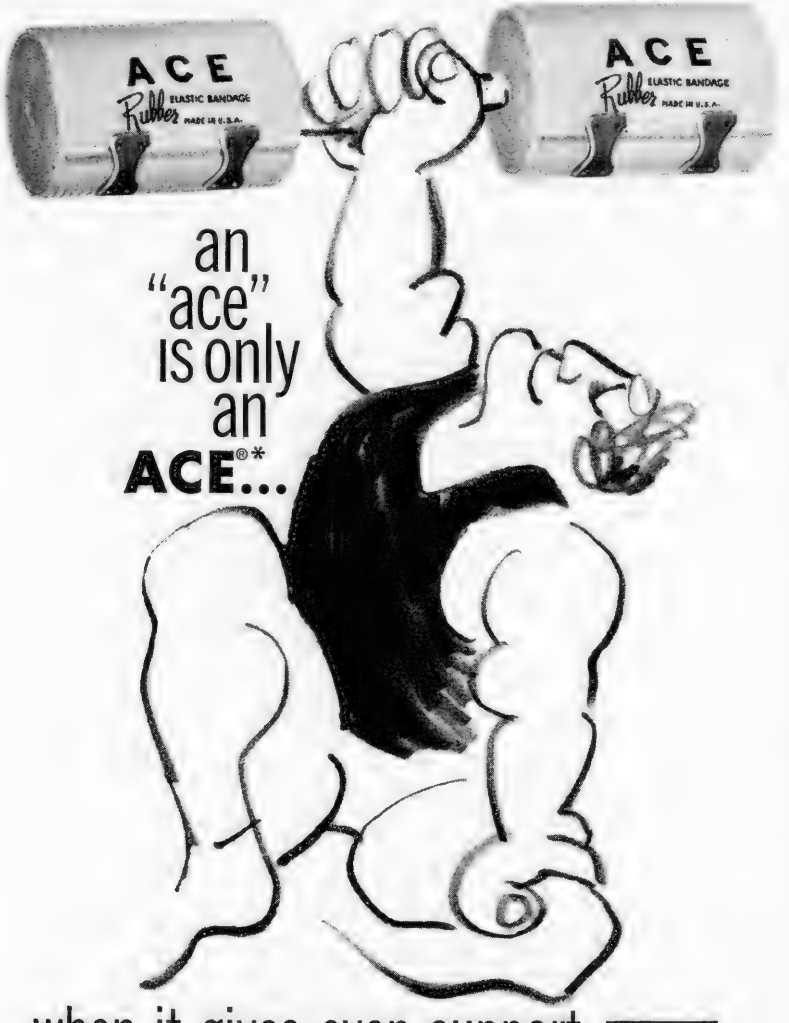
Dr. D'Alonzo holds B.A. and M.D. degrees from the University of Virginia, Charlottesville, Va. After an internship at the Delaware Hospital, Wilmington, in 1938 he pursued training in internal medicine at various medical centers and hospitals. During the war, he was a flight surgeon with the U.S. Army Air Force and later was a section chief at the Fort Bragg Regional Hospital.

Dr. D'Alonzo is a diplomate of the American Board of Internal Medicine and is certified in occupational medicine by the American Board of Preventive Medicine. He is a co-editor (with A. J. Fleming, M.D., and J. A. Zapp, Ph.D.) of the book, "Modern Occupational Medicine" (Lea & Febiger), which had its second edition in 1960. Dr. D'Alonzo is on the Board of Directors of IMA.

► JULES S. CASS, D.V.M., is Assistant Professor of Industrial Health, University of Cincinnati, College of Medicine, Cincinnati, Ohio. He is responsible for health and medical care of the experimental animal colony at Kettering Laboratory, and carries on investigations to ascertain the effects upon laboratory animals of a variety of potentially hazardous chemicals. His twentieth formal publication appears in this issue as "Fluoride: A Critical Review." His preceding publications deal with laboratory animal care, zoonoses, and studies in toxicology.

Dr. Cass received the D.V.M. degree from Ohio State University, Columbus, in 1938. Thereafter he entered graduate work at Ohio and at the University of Minnesota, Minneapolis, Minn., receiving the M.Sc. degree from Ohio; and he took the postgraduate course in the Pathology of Diseases of Laboratory Animals given at Armed Forces Institute of Pathology, Washington, D.C. He joined the faculty at Cincinnati in 1951. He is a member of American Veterinary Medical Association, the Society of Sigma Xi, the American Board of Laboratory Animal Medicine, and the American Board of Veterinary Toxicologists.

—EDITOR



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People and Events

► ROBERT P. SIM, M.D., is the new Medical Director of the Esso Standard Region, Humble Oil and Refining Company, New York City. He succeeds LEO WADE, M.D., Medical Director since 1951, who resigned in June to become Vice-President for Administration for the Sloan-Kettering Institute for Cancer Research.

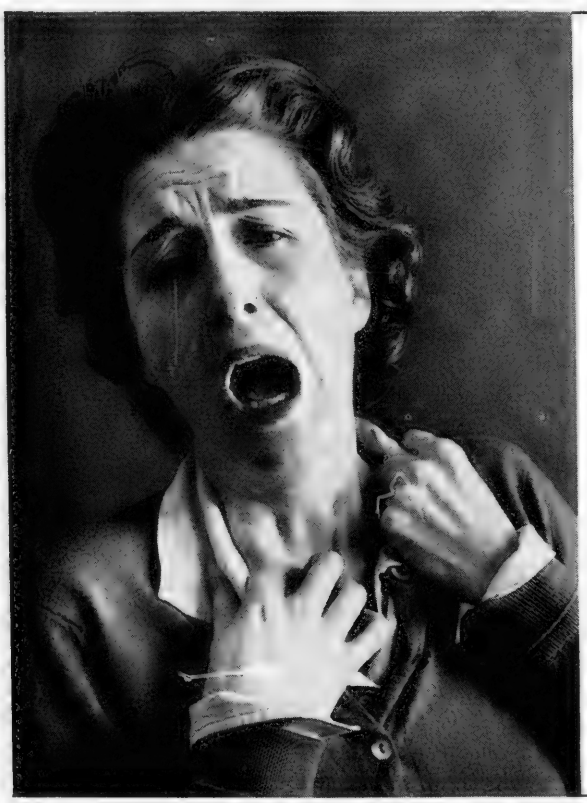
DR. SIM has been Esso's Assistant Medical Director for Marketing since 1953. He received his M.D. degree from Columbia University College of Physicians and Surgeons in 1934. He served his internship at Grasslands Hospital in Valhalla, N.Y., and his residency at Lawrence Hospital in Bronxville, N.Y. From 1937 to 1938 he studied under a fellowship in industrial medicine at DeLamar Institute of Public Health at Columbia University. He joined Standard Oil Company of New Jersey in 1938 and has worked almost entirely since then on the development and administration of medical programs for Esso field marketing units.

DR. SIM received the master of public health degree from Columbia University School of Public Health in 1952. He was certified in public health by the American Board of Preventive Medicine in 1952. He is a member of the American Academy of Occupational Medicine, the American Public Health Association and is a Fellow of the Industrial Medical Association. He is a member of the Executive Committee of the New York State Society of Industrial Medicine.

► CREED F. MCFALL, JR., M.D., has been appointed Assistant to the Medical Director of the American Telephone and Telegraph Company. In this capacity he will assist L. H. WHITNEY, M.D., Medical Director, in the planning and coordination of occupational health programs for the industry.

For the last two years DR. MCFALL has been Medical Director of the Intercontinental Chemical Corporation. Prior to that time he was associated with American Cyanamid Company. He is a graduate of George Washington University, having received his M.D. degree from the School of Medicine in 1948. As a part of his graduate training he served as an associate in pharmacology at the Bowman Gray School of Medicine, Winston-Salem, N.C., a resident in internal medicine at the Queen of Angels Hospital in Los Angeles, and a resident on the chest service (Columbia University), at Bellevue Hospital, New York City.

► WILLIAM L. SHIELDS, M.D., has been appointed Director of Medical Group III of Chrysler Corporation, according to M. W. JOCZ, M.D., Corporate Medical Director. In his new position, DR. SHIELDS directs the activities of medical departments at Center Line, Detroit Tank,



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*Glaser, J.: Ann. Allergy **18**:510 (May) 1960.

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DR. SHIELDS received his M.D. degree from the University of Toronto in 1922 and served his internship at St. Mary's Hospital in Detroit. He joined Chrysler Corporation in 1933. At the time of his present appointment he was senior physician at Chrysler's Highland Park plant. He is a Fellow of the Industrial Medical Association and a member of the Detroit Industrial Physicians club.

► LOGAN T. ROBERTSON, M.D., occupational health consultant, Asheville, N.C., has been named medical consultant to the president of the New York State Civil Service Commission. His duties will involve the development of policies and activation schedules for an occupational health program to serve the 100,000 employees of the New York state government.

DR. ROBERTSON is a Director of the Industrial Medical Association and is a former District Counselor for the state of North Carolina.

► The New York Academy of Medicine inaugurated its newly formed Section on Occupational Medicine at a dinner meeting held in the Presidents' Gallery of the Academy building on September 27. The after-dinner address was presented by JAMES H. STERNER, M.D., Medical Director of Eastman Kodak Company and President of the National Health Council. DR. STERNER'S subject was "The Occupational Physician and the Community."

The Section on Occupational Medicine was formed at the request of a group of Fellows of the Academy who are particularly interested in this field of medicine, and was approved by vote of the Fellows of the Academy and the Academy Council. NORMAN PLUMMER, M.D., and DAVID H. GOLDSTEIN, M.D., are Chairman and Secretary pro-tem, respectively, of the Section.

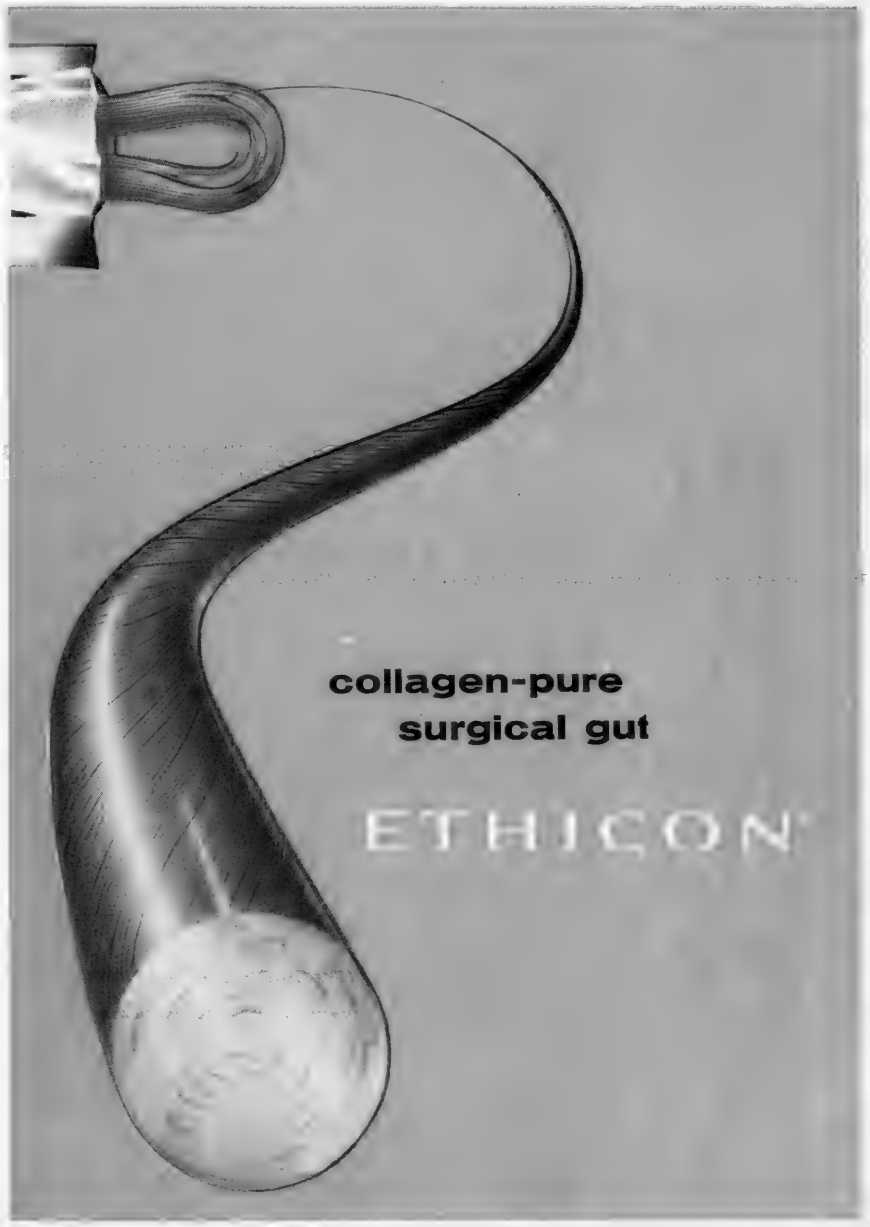
► Industrial Medical Association of Philadelphia will hold its annual President's Dinner on Saturday, November 4 at the Presidential Apartments in Philadelphia.

► The Industrial Medical Administrators Association held its Third Annual meeting in New York City

Deaths

FRANK RAY GRIFFIN, M.D., Toronto, Quebec, on July 5 of accidental drowning at age 63; consultant in occupational medicine for Lever Brothers, Ltd., Canada Metal Co., Ltd., Sun Oil of Canada, Westinghouse Electric Corporation and others; M.D. degree, University of Toronto, 1923; Industrial Medical Association member, 1942, Fellow, 1946, Director, 1946-1948.

HAROLD W. WOUGHTER, M.D., Flint, Mich., on Aug. 16 at age 54; consultant in occupational medicine for several General Motors' plants; M.D. degree, University of Michigan, 1932; Industrial Medical Association member, 1935, Fellow, 1953.



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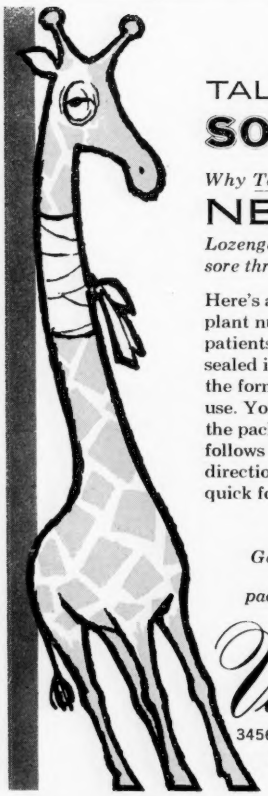
on September 28 and 29. The two days were devoted to visiting the recently-opened medical facilities of New York Telephone Company, Western Electric Company and Equitable Life Assurance Company.

Officers elected for the ensuing year are as follows: President—Tom Sprentall, M.D.; Vice-President—Carl Goutell, M.D.; and Secretary-Treasurer—Richard MacDiarmid, M.D.

► The Fifth Annual Western Industrial Health Conference will be held October 6 and 7 at The Biltmore Hotel in Los Angeles. The meeting is sponsored by the Western Industrial Medical Association, Western Industrial Nurses Association, American Industrial Hygiene Association, American Society of Safety Engineers, American Conference of Governmental Industrial Hygienists, and the Health Physics Society of California in cooperation with the Los Angeles Chamber of Commerce.

One of the features of the program will be a debate on the subject "Resolved: That the Preplace-



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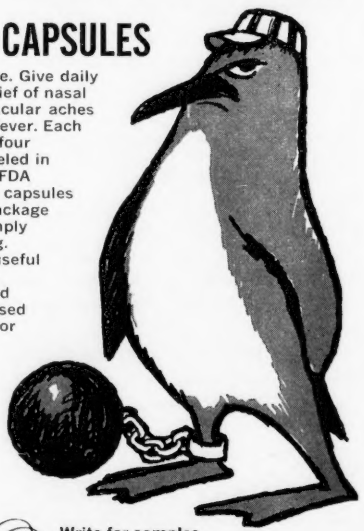

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ment Physical Examination in Industry has Outlived its Usefulness." Other major discussion topics are as follows: "Epidemiology of Accidents"; "Communication—Panacea or Placebo?"; "Toxicology and Occupational Health"; "Are Medical Centers and Hospitals Ready for Employee Health Services?"; "Everyday Medical Care in the Industrial Dispensary"; "Translating Industrial Medical Programs Into Dollars and Cents Values"; "The Employee who Refuses Treatment—Legal Aspects"; "Are There Occupational Health Responsibilities in Reviewing Plans for New Construction?"; "The Newest in Occupational Health Nursing—A View of the 60's"; and "The Realities of Rehabilitation."

► A new syndicated radio series of health education messages entitled "Doctor's House Call" is currently being offered to radio stations throughout the country for local sponsorship. The three-minute taped messages which are being produced in cooperation with the American Medical Association are presented by JAMES R. FOX, M.D., Chairman of the Minnesota Medical Association's Radio-TV Committee.

DR. FOX is an industrial physician for a number of industries in Minneapolis. He has been presenting radio health broadcasts in Minnesota for many years. He is a Fellow of the Industrial Medical Association.

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James B. Workman

SBS Soap Counselor on the Eastern Seaboard, has helped many plants update their wash-up practices. Here's his report after surveying a electrical equipment manufacturing plant:

"We observed the usual powdered soap waste in 23 of the 26 washrooms surveyed. Soap scattered about made an unsightly mess of the washbasins, made the floors dangerously slippery and made cleaning-up twice the job it should have been. You could see the plant's hygiene dollars — both manpower and materials — literally disappearing down the drains!

"To correct this problem, we recommended SBS 60, a thick-bodied cream that clings to skin, won't run-off hands or faces like a powdered soap and is almost impossible to waste. They were convinced when we told them that based upon our experience in other plants, a single gallon of SBS 60 will give their employees 2400 economical, waste-free washes!"

Money-saving suggestions **like this *begin* with OPERATION PINPOINT–a thought-provoking presentation filled with facts about skin hygiene and washroom maintenance. The SBS soap counselor serving your area can pinpoint the right soap to do every skin cleansing job best and at lowest cost in your plant. Let him show you OPERATION PINPOINT . . . just call your nearest SBS office, collect.**

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A Major Advance in the Treatment of Hemorrhage

Despite impressive progress in surgical technic, many situations still arise in which control of bleeding is a problem. For example, bleeding from kidney, brain or liver, or the presistent oozing from severed capillaries and veins as the result of surgical procedure or an accident, may often assume life-threatening proportions.

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SURGICEL Absorbable Hemostat offers one of the most dramatic aids to surgical technic that has become available during the past decade.

Surgicel is available in the following sizes: Knitted Fabric Strips 2" x 14", 4" x 8", 2" x 3"
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More complete literature will be mailed upon request.

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Supply: Scored tablets of 1 mg., 2 mg. and 4 mg. Syrup in 120 cc. bottles, each 5 cc. teaspoonful containing 5.1 mg. triamcinolone diacetate providing 4 mg. triamcinolone.

*Edelstein, A. J.: Pennsylvania M. J. 62:1831 (Dec.) 1959.

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